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MMJ

Maryland Medical Journal

Original Articles

*Colitis induced by non-steroidal
anti-inflammatory drugs*
—15—

*Neurodevelopmental outcome
of extremely low birth weight
infants in Maryland*
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Geriatrics for the Clinician

*Improving the use of
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From the President

MMJ
Maryland Medical Journal

Dear Colleagues:

By the time you receive this issue of the Maryland Medical Journal (MMJ), you should have received your copy of the premier issue of the Med Chi Physician, a new monthly news tabloid that will compliment the MMJ by reporting legislative, regulatory, and medical society news, while returning this magazine to its historical scientific focus.

This change is part of a comprehensive effort at Med Chi to improve our communications with our members. We are committed to the publication of the MMJ, which presents high quality original scientific articles and proceedings of scientific meetings that enhance and compliment Med Chi's core mission of advancing and disseminating scientific knowledge. Because production time for the MMJ is much longer than that of the Med Chi Physician, the new publication will place in your hands non-scientific news and information that affects your practice in a timely and lively format.

In addition to creating the Med Chi Physician, Med Chi is implementing applications for its Internet website (www.medchi.org) that will, for example, afford up-to-the-minute access to the full range of legislative information including text of bills, hearing schedules, and the like. We believe these and other measures will bring you information pertaining to all areas of Med Chi activity in a useful and timely manner. We look forward to your comments on these changes, and to your recommendations for other means of improving communications.

Sincerely

Alex Azar, M.D.

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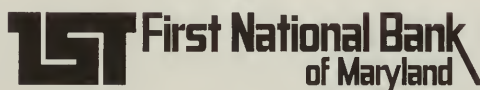


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Obesity Update 1996 fails to convince

In a recent article entitled "Obesity Update 1996,"¹ the author follows four steps to support the position that the approach to obesity needs to be changed. The first is a discussion of the body mass index (BMI) and a suggestion we should use it routinely in the clinic. While it can be used in a clinical setting for the purpose of gathering research data, the BMI has been around a long time with limited clinical utility, that is, assessing patients at the extremes of stature. Try it with your next ten patients. When all is said and done, the patient wants to know "So how much weight do I have to lose?" or "How much should I weigh?" If you like to do the math, you can use the following formula to answer that question: $BMI \times (ht)^2 / 704.5 = wt$. (For a BMI of 25, that reduces to $wt = 0.0355X(ht)^2$. A BMI of 25 corresponds to the new government recommended weights.²)

The second step is an argument for treating obesity. By the author's own admission, "... although there are numerous reasons for a moderately obese patient to lose 20 pounds [note: not reduce BMI from X to Y], it may be disingenuous to claim that the loss will significantly increase longevity." The litany of psychosocial morbidity associated with obesity furnishes no evidence that changing body weight improves the morbid condition. I remain unconvinced.

Step three is a discussion of the genetic and environmental factors in obesity. Certainly, there is exciting research occurring in the genetic and transmitter fields, but no therapy is just over the horizon. I doubt that anyone who prescribes selective serotonin reuptake inhibitors or megestrol believes obesity is just a behavioral problem.

Step four is a discussion of one approach to the pharmacological treatment

of obesity. The author neglects to mention that Weintraub's patients (who were 30% to 80% above Met Life ideal weights with BMIs in the range of 34 to 46) also received behavior therapy, dietary counseling from a dietitian, and a graded exercise program with a goal of an extra 300 kCal three times a week over their usual activity level. The author also chose to cite Weintraub's earliest and most favorable results. In fact, 83 patients were followed for 2 years, with a cumulative 23-pound average loss (only 3 pounds more than the loss at 24 weeks); 48 patients continued to 210 weeks and, on average, were 3 pounds less than when they started the study (7 remained 10% below their start weight, for most, still above their ideal weight).³

Even if the author had convinced me that I should aggressively treat obesity to reduce long-term morbidity and mortality, there doesn't seem to be a successful long-term treatment strategy available. For now, I'll continue to devote my efforts to the more manageable risk factors, and if weight loss occurs as a side effect of treatment, which it often does, so much the better.

RICHARD MOORE, M.D.

Dr. Moore is Director of Public Health Service Health Unit #1 in Bluemont, Virginia.

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2. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 1995;23-24.
3. Weintraub M. et al. Long term weight control study (I-VI, Conclusions), *Clin Pharmacol Ther* 1992;51(5):586-618, 642-646. ■

It is difficult and frustrating to help patients lose weight

Author responds

The respondent raises several important points that highlight how difficult and frustrating it is to help patients lose weight, and which underscore the medical profession's own ambivalence towards the obese. Perhaps the usefulness of the formula for calculating the BMI, like other easily forgotten medical formulae, lies in the eye of the beholder. I myself find it useful to quickly calculate the BMI of new patients so as to accurately compare their degree of overweight with that of other patients I see. The author's second question about whether losing weight would alleviate

the 'psychosocial morbidity' associated is an important one and to my knowledge has not been answered directly. The important point to remember, however, is that much of this psychosocial morbidity comes from how the obese are *viewed by others*, and not from how they view themselves. Thus, it is logical to expect that losing weight will alleviate the 'morbid condition' since the thinner patient will be seen differently by society.

The respondent's last two points concerning the current state of therapy for obesity are essentially accurate; there is no therapy available today for treat-

ing obesity that is as effective as the therapies available for treating other common disorders with both behavioral and biological components such as hypertension, hyperglycemia, or hyperlipidemia. The respondent's reluctance to treat obesity with the currently available pharmacotherapy is not unwarranted, but I take comfort from my sense that the respondent would use pharmacotherapy to treat the medical illness of obesity if such therapy were available in the future.

DAVID UTZSCHNEIDER, M.D., Ph.D.
Dr. Utzschneider practices internal medicine in York, Maine. ■

The sun should not rise or set on a patient with mechanical obstruction

As a surgeon from the antediluvian era, I was entranced by the clarity and definition of the roentgenograms of the abdomen depicted in "Imaging case of the month: Distal small bowel obstruction" (*Med J* 1996;45:558-560). The films are fabulous and repeatedly demonstrate the distal small bowel obstruction. Three modes of films are shown: the plain, the small bowel series following the administration of barium, and the magnetic resonance images.

The history, physical examination, and the preliminary scout films are diagnostic of mechanical obstruction. After proper preparation, including attention to any coexisting medical prob-

lems and the correction of serum electrolyte deficiencies, if they exist, the patient should go to surgery as quickly as possible. Determination of the cause of the obstruction may be attempted primarily, but should actually be left to the time of laparotomy. The prime objective is the relief of the obstruction with attention then being given to its cause.

In my early days, the mortality from the operation for intestinal obstruction was about 50%. With appreciation of the need for fluid and electrolyte replacement, gastrointestinal decompression by tube, use of antibiotics, and better overall surgical treatment, the mortality has dropped to about 10% or

lower. A further reduction of this statistic is quite possible if laparotomy is done in properly prepared patients before simple obstruction progresses to strangulation.

The caveat that the sun should not rise or set on a patient with mechanical obstruction is as true now as it was 50 years ago. This situation is one in which etiology is secondary in importance to relief. Time and money should not be wasted in unnecessary confirmatory tests.

JOSEPH M. MILLER, M.D.
Dr. Miller is a retired surgeon in Timonium, Maryland. ■

The controversial role of imaging in small bowel obstruction

Author responds

The proper role of medical imaging in the scenario of small bowel obstruction is somewhat controversial and has been debated since the introduction of barium in 1897 by Dr. Walter B. Cannon.¹ Numerous imaging modalities have been introduced in the last 100 years, and many of these have applications in identifying small bowel obstruction.

Proponents for increased imaging point out that imaging has been a stabilizing element in our health delivery system and has produced dramatic benefits too abundant to quantify. These individuals also assert that 4% to 5% of health care cost is due to radiology; therefore, the cost-benefit ratio of current imaging techniques justifies their use.

The minimalist view of imaging can probably be justified by considering the time and additional expense wasted in confirming a condition that should be diagnosable clinically. With the dramatic increase in managed care type medical practices, this frugal use of medical services may be potentially rewarded through physician incentives for limited use of resources.

Undoubtedly, the correct use of medical imaging lies somewhere between the two extremes and should be delivered in the proper context of total patient care. The most likely diagnosis in the post-operative patient presented in "Imaging case of the month: Distal small bowel obstruction," should have been obstruction as a result of adhesions. In this setting, a clinical evaluation and plain films would have been

sufficient. If the patient's clinical presentation does not suggest complete obstruction or other complicating factors such as ischemia, a 24-hour trial of nasogastric suction and cautious waiting may be indicated.

If the patient's presentation is atypical for obstruction, concern for other nonoperative etiologies (i.e. some malignancies, bowel motility disorders, or pancreatitis) may lead to further imaging studies.

The choices for further imaging are extensive and must be appropriately chosen for each patient. Patients with a partial small bowel obstruction are probably best evaluated with a barium study,² whether it be a small bowel series or a barium enema (when plain film examination demonstrates a very distal obstruction).

Gross sectional imaging is probably not indicated for complete obstruction unless the surgeon indicates that additional information may alter his surgical approach. Also, Computed Tomography (CT) is best done prior to the administration of barium as this high density contrast agent will degrade the quality of the CT images. Magnetic Resonance Imaging (MRI), using the HASTE technique,³ can provide high resolution multiplanar images even after the addition of barium with scanning times of approximately 10 minutes per patient. The limitations with MRI are the same with all other magnetic resonance examinations (i.e., pacemakers, severe claustrophobia).

The choice of whether to seek further imaging information or whether to

opt for more immediate surgical treatment will undoubtedly continue to be a source of controversy for many years to come. The goal of all involved is to make the appropriate choices that will ultimately give the patient their best chance for a successful and rapid recuperation. The final treatment decision, however, will certainly rest on the physicians' clinical judgment.

Hopefully, the future practicing radiologists and surgeons will not lose sight of the value of good, old-fashioned common sense.

DOUGLAS P. BEALL, M.D.

CARLOS LUGO-OLIVIERI, M.D.

Drs. Beall and Lugo-Olivieri are from the department of radiology and radiological science, The Johns Hopkins Hospital.

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2. Mucha P. Small intestinal obstruction. *Surg Clin North Am* 1987;67: 597-620.
3. Beall DP, Regan F. Technical Note: MR Imaging of Bowel Obstruction Using the HASTE Sequence. *JCAT* 1996 (In Press).

Editorial note: *There must always be concern on the part of the surgeon when he has to operate on an intestine that contains heavy barium (i.e. spillage, peritonitis) which cannot be evaluated because of a partial or almost complete obstruction. The latter can be avoided completely by a small plug of barium.* ■

Stephen T. Bartlett, M.D., John C. Papadimitriou, M.D., Cinthia B. Drachenberg, M.D., David K. Klassen, M.D., Edward W. Hoehn-Saric, M.D., Matthew R. Weir, M.D., and Anthony L. Imbembo, M.D., are among the authors of "Equivalent Success of Simultaneous Pancreas-Kidney and Solitary Pancreas Transplantation." The study, which concludes that modern immunosuppression and biopsy techniques have improved the success of solitary pancreas transplantations, appears in the October 1996 issue of *Annals of Surgery* (224:40-452). The study is from the departments of surgery, medicine, and pathology, University of Maryland School of Medicine.

Lawrence M. Lichstein, M.D., is a contributor to "Clinical Aspects of Allergic Disease: Origin of Latephase Histamine Release." The article is published in the October 1996 issue of *The Journal of Allergy and Clinical Immunology* (98:721-723). Dr. Lichstein is a professor of medicine at Johns Hopkins University.

Carol Carraccio, M.D., Paul Feinberg, M.D., Lisa Sinclair Hart, M.D., Michael Quinn, M.D., James King, M.D., and Richard Lichenstein, M.D., co-authored "Lidocaine for Lumbar Punctures: A Help Not a Hindrance," in *Archives of Pediatrics & Adolescent Medicine* (1996;150:1044-1046). The authors attempt to ascertain whether premedication for lumbar puncture (LP) with lidocaine hinders collection of cerebrospinal fluid (CSF) through either increased number of attempts or increased incidence of traumatic punctures. Drs. Carraccio, Hart, Quinn, King, and Lichenstein are from the department of pediatrics, University of Maryland School of Medicine, and Dr. Feinberg is with The Pediatric Center Community Practice, Frederick.

Moyses Szklo, M.D., DrPH, and Millicent Higgins, M.D., DrPH, are among the authors of an article in the October 15, 1996 issue of *Circulation* (94:1857-1863) entitled "No Association of Menopause and Hormone Replacement Therapy with Carotid Artery Intima-Media Thickness." The facts that cardiovascular disease is the major cause of death in older women, and that information on the relation of menopause and hormone replacement therapy with carotid atherosclerosis is limited, form the basis for the article. Dr. Szklo is affiliated with The Johns Hopkins School of Hygiene and Public Health. Dr. Higgins is associated with the division of epidemiology and clinical applications, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda.

Mark A Klebanoff, M.D., MPH, is one of the authors of "Recurrent Respiratory Distress Syndrome in Successive Preterm Pregnancies." The article concludes that "preterm infants born to women with a previous preterm infant affected by (respiratory distress syndrome) RDS are at an increased risk of RDS, which suggests an important genetic (or other familial) tendency in its origin (*J Pediatr* 1996;129:591-596). Dr. Klebanoff is with the National Institute of Child Health and Human Development, Bethesda.

Ba D. Nguyen, M.D., and A. Cahid Civelek, M.D., coauthor "Bone Scintigraphy of Caudal Regression Syndrome," which is published in the October 1996 issue of *Clinical Nuclear Medicine* (21:802-804). Dr. Nguyen is associated with the department of radiology at Johns Hopkins Hospital, Baltimore. Dr. Civelek is an associate professor of radiology at Johns Hopkins University, and the director of the nuclear cardiology section at Johns Hopkins Medical Institute.

Speak Out

Capitation: A moral issue in the managed care paradigm

Answer the following two questions: Does capitation, as a method for financing managed care plans, ultimately place the best interests of the treating physician and the best interests of the patient in conflict? Doesn't the medical profession, which should function as an uncompromised force for patient advocacy, have a moral responsibility to keep these two interests aligned?

Notice we have not implied that managed care is the villain here, but rather that the concept of capitation, as it is being played out by the insurance driven model, is the antithesis of what the profession and the public have long considered to be right. The public has traditionally revered the Marcus Welby-type physician who performs a personal service and is paid a reasonable fee for taking good care of someone. In the media we do not see a heroic physician being rewarded by an insurance corporation for not providing health care services and not spending the corporation's dollars. Capitation, even when applied by a physician-controlled managed care program, risks limiting services to those patients who need them the most — the sick.

Market force dynamics and intense competition for contracts dictate that price will determine the success or failure of any plan. The race to the cheapest has already taken quality-of-care issues out of our profession (where they belong) and placed them on the table of public relations and advertising agencies for selling to the public. The insurance industry should not be defining medical care, setting standards, deciding on admission to the hospital and lengths of stay. The physician should be making these decisions in partnership with the patient and in collaboration with medical colleagues. If capitation places a tension between the doctor and the patient, which has the potential for harm, the profession should limit it and advocate a reasonable remedy. There should be a way to fairly reimburse physicians for providing reasonable and needed care at a predictable cost without sacrificing the traditional doctor-patient relationship.

We suggest that Med Chi bring together an expert panel to discuss the issue of capitation as a moral dilemma at the next annual meeting. From this dialogue, we hope there is sufficient agreement to develop a resolution to be considered at the House of Delegates. If any component would like to help develop a resolution on this subject, we would like to hear from you.

It has been said that our profession is about to become relegated to the role of mid-level employees of the insurance industry. We are late in managing managed care. Now is the time for us to delineate health care in the context of what is right for our patients and to be uncompromising in that advocacy.

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Colitis induced by non-steroidal anti-inflammatory drugs

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Introduction

Non-steroidal anti-inflammatory drugs (NSAIDs) are widely prescribed; over 100 million prescriptions are written each year. Compared to steroids and narcotic analgesics, NSAIDs have a wide safety margin, but there is growing concern about side-effects, especially as related to the gastrointestinal tract. The upper tract is most commonly affected, but effects on the small intestine and colon are increasingly recognized. Described below are two cases of large bowel ulcers causally associated with ingestion of NSAIDs. In both cases, the patients responded well when the medication was discontinued and recovered without complications.

Case Reports

Case 1

A 64-year-old male was admitted to the hospital with complaints of passing fresh blood per rectum for three hours. He denied abdominal pain, diarrhea, nausea, and vomiting.

He had a hemicolectomy for a villous adenoma nine years ago. He had a history of hypertension and chronic arthritis, and he had taken Indomethacin-SR 75 mg twice daily for two months.

On physical examination, his abdomen was unremarkable, but a rectal exam revealed fresh blood. The hemoglobin level was 12.5 g/dl with hematocrit of 37 %. White blood cell count, SMA-7, and SMA-12 were normal. The patient continued to bleed with a resultant fall in hemoglobin to 8.2 g/dl and hematocrit to 24.9 %. He was given two units of packed red blood cells (PRBCs), and Indomethacin was discontinued.

Colonoscopy revealed the presence of blood from the rectum to about 90 cms. No blood was seen beyond that point. At 90 cms, there was a firm, ulcerated lesion about 2.5 cms in diameter with the surrounding mucosa

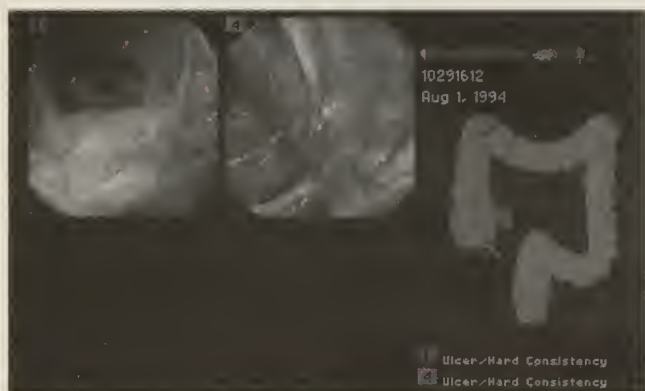


Figure 1.



Figure 2.

appearing normal (Figure 1). Multiple biopsies were taken. A small non-bleeding polyp was noticed at 50 cms. No other source of bleeding was present.

With supportive measures, the patient stopped bleeding and the hematocrit rose to 32.1 %. Biopsy revealed mild non-specific chronic inflammation with no atypia. The patient was discharged.

Repeat colonoscopy performed six weeks later revealed complete healing of the transverse colon ulcer (Figure 2). Diagnosis was made of NSAID (Indocin)-related colon ulcer, which responded to cessation of Indocin, suggesting a causal relationship.

Case 2

A 58-year-old white female presented to the emergency room with complaints of passing fresh blood per rectum for six hours. She had three loose bloody bowel motions. Abdominal pain, nausea, vomiting, and dizziness were denied. She had arthritis of the knee, for which she had undergone surgery and taken Oxaprozin (Daypro) two tablets daily for six months.

On admission, her abdominal examination was benign, but her rectal examination showed fresh blood. Admission

laboratory studies revealed hemoglobin of 11.0 g/dl and hematocrit of 34.3 %, with the rest of her complete blood count, SMA-6, and SMA-12 normal. Over the next 24 hours, her hemoglobin and hematocrit fell to 8.9 g/dl and 27.2 %. She was continued on supportive measures and scheduled for a colonoscopy. Her Oxaprozin was discontinued.

Colonoscopy revealed multiple non-bleeding diverticulae and the presence of focal colitis in the sigmoid and descending colon, with superficial ulceration and erythema (Figure 3). Multiple biopsies were taken and histology revealed edema and mild acute or chronic inflammation consistent with acute ulceration. The patient stopped bleeding and was discharged.

She had a rapid recovery and underwent follow-up flexible sigmoidoscopy four weeks later, which revealed a completely normal sigmoid and descending colon with no residual lesion (Figure 4).

Discussion

The cases described above highlight the adverse effects of NSAIDs on the lower gastrointestinal tract. According to various case reports,¹⁻⁹ colon involvement with NSAIDs can be described under two major groups. One is their effect on



Figure 3.



Figure 4.

normal colon (de-novo colitis), and the other is their effect on pre-existing disease.

De-novo colitis associated with NSAIDs has been described in the literature.¹⁰ The most common age group described is the seventh decade with duration of exposure varying from two days to twelve years, with a median of three months. A relatively large number of cases are due to fenamates (mefenamic and flufenamic acid), Diclofenac, and Indomethacin, probably a consequence of excessive use. The most common presentation is bloody diarrhea with weight loss and anemia. Colonoscopy may be normal, or may reveal inflammation and ulceration resembling ulcerative colitis, with the extent of disease varying from proctitis to pancolitis. Histology mostly shows non-specific inflammation and lymphoid infiltration. On discontinuing the drug, complete symptomatic and histological recovery has been noticed. There are also reports of ulceration of the cecum, transverse, and sigmoid colon.^{9,11} In a large retrospective study by Langman, et al., of 268 patients with colonic or small bowel perforation or hemorrhage, patients with perforation or hemorrhage were twice as likely to have taken NSAIDs.¹²

Other forms of damage to normal colon have been seen in the form of collagenous colitis, eosinophilic colitis, and pseudomembranous colitis.⁵⁻⁷

NSAID-induced colitis is under-diagnosed. Lower gastrointestinal bleeding, especially in the elderly, is often labeled idiopathic, or an assumption is made that the bleeding was from diverticulosis, ischemia, or angiodysplasia, although no specific site is identified. In 1988, Tanner and Raghunath estimated that 10 % of newly diagnosed colitis may be related to NSAIDs, and that patients taking NSAIDs were five times as likely as the general population to develop colonic inflammation.³

Effects of NSAIDs on pre-existing disease have been described in the form of reactivation of quiescent inflammatory bowel disease^{13,14} and hypersensitivity colitis to salicylates.⁴ Proctitis resulting from NSAID suppositories, especially Indomethacin, has also been reported.¹⁵

The mechanism of NSAID-related colitis may relate to the inhibition of intestinal prostaglandin synthesis, and also diversion of arachidonic acid toward the lipoxygenase pathway, producing leukotrienes and free oxygen radicals causing inflammation and tissue injury in the gut.¹⁶ Phillips and Hoult studied transepithelial potential difference, a measure of membrane integrity, and found that Indomethacin and other NSAIDs reduced this potential, and reduced prostaglandin E2 production from cells of lamina propria.¹⁷ Bannerjee and Peters studied the effect of Indomethacin on rat intestine and showed enhanced mucosal permeability, increased tissue myeloperoxidase levels, and reduced weight gain analogous to inflammatory bowel disease in the human.¹⁸

Conclusion

Diagnosis of NSAID-induced colitis requires increased awareness and suspicion that the problem exists. NSAID-induced colitis should be considered in the differential diagnosis of abdominal pain, diarrhea, bleeding per rectum, unexplained anemia, weight loss, and signs or symptoms of inflammatory bowel disease. An effort should be made to limit the total dose of NSAIDs, with particular attention to elderly patients. Caution should be exercised in prescribing for patients with inflammatory bowel disease.

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Neurodevelopmental outcome of extremely low birth weight infants in Maryland

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ABSTRACT: *The survival rate of extremely low birth weight (ELBW; i.e. <1001 grams) infants has significantly improved in the past 10 years secondary to the numerous advances in neonatology. There have been many favorable reports of the neurodevelopmental outcomes of survivors, but the studies often span several years to collect sufficient number of subjects. This study assesses the outcome of 100 ELBW infants born in Maryland in 1990 and analyzes factors that may have contributed to their outcomes at one year corrected age.*

Of this group, 72% had no evidence of severe disability (e.g., cerebral palsy (CP) or mental retardation (MR)); however, 51% of the children had abnormal or suspect neurological examinations, and 24% had CP. Eighteen percent of the children were more than one standard deviation below the mean cognitively; 30% were below normal for motor abilities, and 33% were below normal for language abilities. Prior to this study, many of these children were not recognized by their primary physician as having any developmental problems. Many of these children were not followed in neonatal intensive care unit (NICU) follow-up programs, and most were not receiving appropriate early intervention services (EIS).

Previous studies have associated different neonatal events with the risk of developmental delay. Bronchopulmonary dysplasia (BPD) and periventricular leukomalacia (PVL) accounted for most of the variance of this sample's developmental outcome. Of these 100 ELBW infants, 56 received surfactant. Analysis demonstrated no significant differences in developmental outcomes between those who received surfactant and those who did not. However, those who received

rescue surfactant were more likely to acquire a diagnosis of BPD.

As demonstrated by this study, ELBW infants are at risk for significant developmental problems. This supports the need for targeted outreach, developmental monitoring, early intervention services, and parent support and education.

Introduction

Maryland has a history of producing more low birth weight babies than the national average.¹ One hundred and two infants weighing under 1001 grams (ELBW), born from January through August, 1990, were studied to evaluate their neurodevelopmental outcomes. This study population was drawn from an ongoing study, begun in 1989, to evaluate Maryland women with regards to the risk factors associated with preterm delivery.

The surviving ELBW infant is at high risk for many neonatal complications secondary to his/her immaturity, frequently resulting in developmental disabilities, such as CP and MR. While the rates of severe disabilities have not changed, the survival rate has greatly improved. In 1960, mortality was 92%² for ELBW infants; this has greatly decreased to 23% in 1990. Reported rates of MR range from 7% to 27%,³⁻⁵ while the rate of one or more major neurodevelopmental impairments is approximately 25%.^{6,7} Previous research in the area of developmental outcome has linked the presence of disabilities to only a few perinatal complications, the most prominent being pulmonary disease and intracranial abnormalities.

Pulmonary disease is still one of the most significant problems for ELBW infants. Treatment with surfactant has been shown to affect the morbidity and mortality due to respiratory distress syndrome (RDS) by decreasing the incidence of death or prolonged need for oxygen. Some studies of surfactant use have noted decreased incidence of pneumothorax (PTX) and intraventricular hemorrhage (IVH).⁸⁻¹¹ Surfactant has also been shown to have its greatest effects on infants less than 30 weeks gestation⁷ or less than 1001 grams,¹² that is, the ELBW infant. Because BPD and IVH are perinatal risk factors for poor neurodevelopmental outcome,¹³⁻¹⁵ it may be hypothesized that surfactant may decrease the incidence of poor neurodevelopmental outcome in these ELBW infants.

In eight studies of the effects of surfactant on the health and developmental follow-up of premature infants, no statistically significant differences were found between those who received surfactant and those who did not.¹⁶⁻²³ One of the largest studies had 76 subjects, 45 surfactant treated infants and 31 controls evaluated at one year corrected age.

Yet, only 33 of the 76 children were completely assessed with regards to neurological and developmental outcomes.¹⁶ The largest study available consisted of 80 surviving infants followed to two years corrected age, but these children were <34 weeks gestation, with the mean birth weight being 1569 grams for the surfactant group and 1672 grams for the non-surfactant group. No language assessment was performed.²³

Because surfactant had not been approved by the Food and Drug Administration prior to the birth of any of the subjects in the present study, all infants who received surfactant did so under a research protocol; therefore, the chance of receiving surfactant depended on where the baby was born (or transferred to) and when that hospital began a research protocol. In Maryland, the surfactants in research protocols in 1990 were Survanta, Exosurf, and Infracurf. No difference in effectiveness or outcome has been found for the different surfactants. Other studies have compared the use of prophylactic to rescue surfactant with conflicting results.²⁴⁻²⁶

This study looks at the neurodevelopmental outcomes of ELBW infants, investigates the factors that contributed to their development, and identifies unmet needs with regards to follow-up and early intervention services.

Methods

Subjects

All subjects were ELBW premature infants (weighing less than 1001 grams), born between January 1, 1990, and August 31, 1990, whose mothers were residents of Maryland. Hospitals in Maryland notified one of the investigators at the time of an ELBW infant's birth. The study was approved by the institutional review board at each hospital.

Seven hundred and seven ELBW infants were born to Maryland residents in 1990. Of the 248 infants referred for this study, 191 survived (a 77% survival rate) to hospital discharge. Eighty-nine possible subjects declined participation or did not appear for their appointments. The only information available about the nonparticipating infants was their birth weight. One hundred and two subjects (19% of Maryland's surviving ELBW infant population) were assessed at one year corrected age.

Information regarding the presence of meningitis, PVL, hydrocephalus, Grades 3 or 4 IVH (as defined by hospital discharge summary), seizures, BPD, days on oxygen, and discharge medication were recorded from the hospital discharge record after all subjects were tested.

Exclusion criteria included the presence of serious congenital malformations, genetic disorders, diagnosed congenital infections, meningitis after discharge from the NICU, congenital syndromes with diagnosed brain abnormality or known correlation with developmental disability, disorders secondary to prenatal toxic exposure with micro-

Table 1. Infant characteristics

Characteristics	Percentage or Mean +/- SD
Female	64.0
Non-white	60.0
Gestational age (weeks)	26.5 +/- 2.4
Birth weight (gms)	775.9 +/- 138.4
Head circumference (cms)	45.2 +/- 1.5
Corrected age (mos)	12.0 +/- 0.6
Inborn	73.0
Appropriate for gestational age	76.0

cephaly, and severe head injury after discharge. Two infants were excluded from the analysis because they developed meningitis after discharge from the initial hospitalization. This decreased the total study population from 102 to 100.

Procedures

Parental Interview. Each subject's parent (usually mother) was interviewed at the time of the infant's neurodevelopmental evaluation at one year corrected age (+/- 1 month). The sites for interview and evaluation included NICU follow-up clinics, pediatricians' offices, and subjects' homes. Information collected regarding the infant included interim medical history, developmental milestones, and demographic data.

Information regarding mother's date of birth, years of education, marital status, home address, phone number, race, and monthly household income was also obtained. Income per month was divided into five categories: 1 = <1000, 2 = 1000 to 1500, 3 = 1500 to 3000, 4 = 3000 to 5000, 5 = >5000. The parent was asked to choose a category into which his/her family presently fit. Marital status was classified as married, single, divorced, or other. The presence or absence of early intervention services was also obtained at interview. The presence of prenatal care was recorded as positive if the parent reported it during the interview or if it was noted during later chart review. The use of illicit drugs was documented in the same way.

The evaluator was blinded to subject's neonatal course, including treatment with surfactant. The parents were asked not to reveal this information to the evaluator.

Developmental Evaluation. Each infant was evaluated by a developmental pediatrician at 12 months corrected age (+/- 1 month). The Bayley Scales of Infant Development (BSID),²⁷ mental developmental index (MDI), psychomotor developmental index (PDI), and the Clinical Linguistic and

Auditory Milestone Scale (CLAMS)²⁸⁻³⁰ were used for evaluation of the infant's developmental status. A developmental quotient (score in months/age) was calculated for the CLAMS. Receptive and expressive language subscores for each infant were also calculated and recorded. All developmental quotients (MDI, PDI, and CLAMS) were corrected for prematurity using the parent's reported gestational age of the infant.

Neurological Evaluation. A standard neurodevelopmental examination, modified after Amiel-Tison³¹ and the Primitive Reflex Profile,^{32,33} along with measurement of the infant's head circumference, was performed by a developmental pediatrician.

Results of the neurodevelopmental examination were recorded as presence of CP, absence of CP, or "suspect." CP was defined as abnormally increased tone in at least one extremity associated with increased deep tendon reflexes, plantar extensor responses in the affected lower limb, and significantly delayed motor development. Subjects who had only mild motor delay and/or decreased or increased tone, without clonus were classified as "suspect."

Statistical Analysis

Pearson's correlation coefficient and step-wise, hierarchical multiple regressions were used to evaluate predictors of developmental outcomes. Univariate analyses (chi square and student's two-tailed t-tests) were used to determine if there were any differences between those who received surfactant and those who did not.

Results

Sociodemographic data

Characteristics of the infants participating in the study are shown in **Table 1**. Maternal characteristics are in **Table 2**. Most of the mothers were not teenagers and had at least a high school education.

Further demographic data was obtained from the Maryland birth certificate registry. The participants' mothers in

Table 2. Maternal Characteristics

Characteristics	Mean +/- SD; percentage
Age (years)	29.8 +/- 6.7
Education (years)	13.1 +/- 2.7
Income per month ¹	2.98
Prenatal Care	92.0
Substance Abuse	10.0
Marital Status ²	59.0

¹categories described in methods section

²percent married

this study were comparable to the mothers of all ELBW infants in Maryland in 1990 with regards to race (40% of the women in this study were white versus 31.4% in Maryland in 1990), education (39% had 12 years of education versus 40%), age (64% were between 20 and 34 years old versus 73%), and marital status (59% were married versus 45.7%). Income data was not available from the birth registry.

Medical History

Table 3 shows the various hospital complications of study infants including the presence of meningitis, PVL, hydrocephalus, seizures, IVH, BPD, retinopathy of prematurity (ROP), and whether the infant was discharged on oxygen. BPD was very frequent (63% incidence), while one-quarter of the infants had grade 3 or 4 IVH.

The mean birth weight of the 100 participating infants ($m=775.9$ gms ± 138.4) was compared to the mean birth weight of the 89 potential subjects who declined participation ($m=814.1$ gms ± 119.5) and was not found to be significantly different. Access to the medical records of the 89 non-participating infants was not available for further study; therefore, other medical complications and whether they influenced participation in this study were unable to be determined.

Developmental Outcome

Table 4 shows the mean scores obtained from the developmental assessments. Eighty-two percent and 70% of the infants had MDIs and PDIs, respectively, within one standard deviation from the mean (>84). Nine percent of the MDIs and 12% of the PDIs were between 70 and 84 (between one and two standard deviations). Abnormal results (<70) were present on 9% on the MDIs and 18% on the PDIs of all subjects. By definition, only 3% of the general population score less than two standard deviations from the mean, demonstrating a substantially higher percentage of ELBW infants in the significantly delayed range.

Table 3. Hospital Course

Complications	Percentage
Meningitis	6
Periventricular leukomalacia	10
Hydrocephalus	18
Seizures	7
Intraventricular hemorrhage (grade 3 or 4)	26
Bronchopulmonary dysplasia	63
Discharged on oxygen	20
Retinopathy of prematurity	49

In comparison, only 66% of the total sample had language skills within one standard deviation from the mean. Thirty-four percent of the children were greater than one standard deviation below the mean (compared to an expected 13% of the general population), and 12% of the sample had significantly delayed total language skills, greater than two standard deviations below the mean (compared to 3% of the general population). Most of these children also had significantly delayed cognitive abilities, but 3% of the total sample had language delay that was not associated with abnormal MDIs or PDIs.

Two-tailed, paired t-tests were done to assess the differences between expressive and receptive language scores. Overall, the ELBW infants had better receptive than expressive language skills (CLAMS-R = 99.72 ± 21.3 ; CLAMS-E = 90.79 ± 26.2 ; $T=6.31$; $p<.001$) (**Table 4**).

Multiple regression analysis was performed to determine if any variables had a significant effect on the developmental outcomes of the infants. Within this ELBW group, the birth weight did not relate to developmental outcome. Similarly, treatment with surfactant did not affect developmental outcome. In contrast, PVL and BPD accounted for most of the increased chance of impairment. PVL, BPD, seizures, and hydrocephalus accounted for 29.7% of the variance on the MDI scores ($F=10.05$, $p<.001$). For the PDI scores, 23% of the variance was accounted for by PVL, IVH, and BPD ($F=9.6$, $p<.001$). For the total CLAMS score, 11% of the variance was accounted for by PVL and BPD ($F=7.1$, $p<.002$).

Neurological Outcome

The neurodevelopmental examination revealed the presence of CP in 24% of total subjects; 49% had normal examinations and 27% had suspect exams. Regression analysis was performed to determine which variables had a significant effect upon the neurological outcome. Results showed that within this ELBW group, lower birth weight did not increase the risk of neurological impairment. In contrast, BPD and PVL increased this risk (14.8% of the variance; $F=8.4$, $p<.001$); the presence of IVH may have contributed as well ($p=.06$). No other individual variables approached significance.

Sensory Impairment

Hearing loss was noted in 16 of the 100 subjects, with only one child wearing a hearing aide. None of the children were profoundly hearing-impaired.

Vision problems were defined as strabismus, hyperopia, myopia, or retinal detachment. Eighteen subjects had vision problems; of those, two had retinal detachment. Three children wore glasses. None were clinically blind.

Surfactant: Rescue and Prophylactic

As noted previously, 56 infants in the sample received surfactant. There were no statistically significant differ-

ences in neurodevelopmental outcomes between the children who received surfactant and those who did not. Thirty-nine (69.6%) received prophylactic and 17 (30.4%) received rescue surfactant. Using chi-square analysis, a significant difference was found with respect to BPD; the infants who received rescue surfactant were more likely to acquire a diagnosis of BPD ($p=.03$). No differences were observed between children who received rescue and prophylactic surfactant in developmental outcome, neurologic examination, or other events during the hospital course.

Early Intervention Services

Maryland has provided early intervention services (EIS) to very young children since 1980. In 1992, The Individual with Disabilities Education Act was established, thus continuing federal funds to provide EIS to children with developmental delays, birth to three years of age, in a family-friendly manner. Maryland established that any child was eligible for services from the Maryland Infant and Toddler Program if he/she had: 1) a 25% delay in any developmental area; 2) atypical development or behavior which is demonstrated by abnormal quality of performance and function in any developmental area; or 3) a physical or mental condition that has a high probability of resulting in developmental delay (i.e. a birth weight of less than 1200 grams). Therefore, all of the infants in this study were eligible. However, only 17% of the parents remembered hearing of the program, and only 31% were receiving EIS.

Discussion

This study, as a descriptive analysis of ELBW infants in Maryland, supports the findings of previous studies indicating that ELBW infants are at higher risk for developmental problems than their full-term and larger preterm peers. However, only 31% of the children in this study were receiving EIS; many more than 31% were detected as having developmental problems at one year corrected age. Primary care providers need to be aware of this risk and the range of disabilities that this population may represent, both in variety and severity. The primary care provider needs to provide careful and repeated developmental surveillance and work collaboratively with NICU follow-up programs and the local infant and toddler programs. This will enable the practitioner to provide earlier detection and earlier intervention for these high risk children.

All of the children in this study were eligible for EIS under the high probability criteria of the Maryland Infant and Toddler Program (birth weight of less than 1200 grams). However, most of the children in this sample were not detected as having developmental abnormalities despite their known risk status. Moreover, parents were not aware

Table 4. Developmental Quotients¹

Developmental Measure	Mean +/- SD
MDI ²	99.7 +/- 19.8
PDI ³	88.9 +/- 19.3
CLAMS-T ⁴	94.9 +/- 23.0
CLAMS-E ⁵	90.8 +/- 26.2
CLAMS-R ⁶	99.7 +/- 21.3

¹Corrected for prematurity

²MDI= mental developmental index

³PDI= psychomotor developmental index

⁴CLAMS-T= Clinical Linguistic and Auditory Milestone Scale-total

⁵CLAMS-E= Clinical Linguistic and Auditory Milestone Scale-expressive

⁶CLAMS-R= Clinical Linguistic and Auditory Milestone Scale-receptive

of this available resource for parent support, developmental monitoring, and family-focused services. The reasons for this are unclear, but may relate to parents being overwhelmed at time of discharge and/or the relative newness, at the time, of the Infant and Toddler Program.

Only 26% of the children at one year corrected age were totally normal on every aspect of examination. Eighteen percent of the cognitive assessments were below normal. Sixteen percent of the children had hearing loss and 18% had vision problems; none of the children had complete loss of vision or hearing. Language was delayed in 34% of the children. CP was diagnosed in 24% of the subjects, but only 49% had completely normal neurological examinations.

Halsey's report³⁴ supports this study's findings. Seventy-seven percent of the ELBW children in his study were without severe disability; only 26% obtained at least average scores on all measures of development. Halsey's study was based upon a smaller sample size and was predominantly white, middle class children. Moreover, in Halsey's study, children who scored in the average to above-average range in cognitive testing were noted to have very uneven test profiles with below-average scores in language, visual-motor integration, and fine/gross motor.

Language appeared to be the ELBW infants' greatest area of weakness; only 66% of the sample had age-appropriate language skills, and expressive language was often more delayed than receptive language skills. Klebanov, et al., report that these ELBW children demonstrate lower attention and language skills, with overall lower social and scholastic competence,³⁵ placing them at an overall higher risk for learning disabilities. The present study noted three children without significantly delayed cognitive skills and without CP, but with significantly delayed language skills. Further study of these children,

at older ages, is recommended to evaluate for future language-based learning disabilities.

Byrne, et al., found an even higher percentage of expressive language delay when low birth weight (<1500 grams) infants were assessed at 24 months, as compared to 12 months (28% versus 8%, respectively).³⁶ Later assessment may document an even higher percent of language delay in our population. Detection of delayed language is therefore critical to later school success.

As in most studies, not all potentially eligible subjects were studied; 89 possible subjects declined follow-up or could not be reached. In order to determine if the two groups were comparable, mean birth weight was evaluated and found not to be different between the groups. Therefore, we do not believe our sample was biased for birth weight, but it is possible that the participating infants may have represented families with more concerns regarding their child's developmental status. Other attempts to determine if selection bias was operating were inconclusive. However, the outcomes reported in this study are similar to other studies, despite possible selection bias.

In this group of ELBW infants, birth weight within the group did not seem to be related to neurodevelopmental outcome. This is in contrast to other studies of developmental outcome which find birth weight a predictor. This may be because these children were all ELBW infants, and therefore, differences within this group were not demonstrated. An alternative hypothesis is that our sample size was not large enough to demonstrate a difference.

Because environmental factors may strongly influence a child's developmental outcome, sociodemographic characteristics were compared and found to be similar to 1990 Maryland demographic data. This indicates that the higher percentage of developmental abnormalities is likely due to ELBW, not socioeconomic status or other demographic factors.

Many studies have now demonstrated the effectiveness of surfactant in reducing short-term morbidity and mortality due to RDS. Earlier studies that included later developmental outcomes have had smaller sample sizes and/or have looked at infants with higher birth weights than this current study of 100 ELBW infants. The results of this study confirm pulmonary disease and intracranial events are the most significant contributors to developmental outcomes in ELBW infants. However, there were no developmental differences with regards to treatment with surfactant, despite surfactant's demonstrated beneficial short-term effects on the respiratory status of newborns.

The fact that intraventricular hemorrhages continue to be a major contributor to developmental problems further sup-

ports the need to continue to work towards decreasing the incidence of IVH.

In conclusion, we found that BPD and PVL were the most significant contributors to future neurodevelopmental outcomes, and that most children, despite their extremely low birth weight, were doing fairly well developmentally. While many of the ELBW infants had positive outcomes with respect to measurement of cognitive skills, language skills, and neurodevelopment, there is still a significant number of children who are not within normal limits on any of a variety of developmental assessment tools. Even those infants who are normal at one year corrected age are still at risk for later learning problems. Early intervention for assessment and provision of services is warranted, because it may decrease the frequency and severity of delays in development. A review of the effectiveness of early intervention programs by Simeonsson, Cooper, and Scheiner³⁷ found that 81% of the studies that incorporated statistical procedures yielded statistical evidence for effectiveness. In 93% of the studies the authors concluded effectiveness, but they may not have had statistical evidence to support this due to inability to quantify some results.

Third party payors need to be aware of the special needs of these ELBW infants, in order to support collaborative care between their primary care providers and developmental follow-up by qualified individuals (i.e., developmental pediatricians, occupational and physical therapists, speech therapists, social workers, nurses, nutritionists, and early child developmental specialists) in a regular and systematic fashion. In this way, the large investment that has been made in these children's medical care can produce the best neurodevelopmental outcome for the child and family.

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Geriatrics for the Clinician

IMPROVING THE USE OF ADVANCE DIRECTIVES

Preventive health is a major component of geriatric medicine. While immunizations and other traditional preventive practices have become standard care, the same diligence is not demonstrated in the use of another form of preventive care — advance directives. Despite widespread support in principle, many physicians do not routinely discuss advance medical planning with their patients. As a result, some patients receive unwanted or inappropriate medical care. This article presents a case for greater use of advance directives and describes a process for establishing these instruments.

Barriers and benefits to advance directives

Physician oversight in the use of advance directives occurs for several reasons. Most often it is due to lack of knowledge and experience in advance medical planning. Additional explanations include attitudes about who benefits from such planning, discomfort in discussing end of life issues, and time demands.¹

Physicians fear that end of life discussions cause patients to become anxious or depressed. In fact, most elderly patients have considered life-sustaining treatment and surrogate decision makers. Numerous studies show that patients wish to have such discussions with their physicians.² Even critically ill patients desired discussion of resuscitation and life support according to the SUPPORT study.³ (The support study attempted to improve end-of-life decision making through the use of a specially trained nurse who facilitated discussion between patient, family and physician.) In general, older patients fear loss of dignity and control more than death.

The value of advance medical planning in the elderly is apparent. They are by far the most likely to experience life-threatening illness. Such events may render them unable to communicate. However, all ages are vulnerable to catastrophic illness,

and prior discussion can obviate much uncertainty. Two landmark cases, Karen Quinlan and Nancy Cruzan, involve termination of life support in younger patients. For these reasons, discussion of advance directives is recommended for all ages. A compelling argument is made for including advance directives as part of routine preventive health. Such discussions help cement the therapeutic alliance between patient and physician by reassuring that the physician will act as advocate. This advocacy continues in the event of incompetence or critical illness.

Linda Emanuel, M.D., Ph.D., and colleagues describe the value of “process” in developing the medical directive. She describes a process of advance care planning that is worth reading.⁴ This process need not be accomplished during a single consultation and for our purposes will be divided into three steps. These steps include introduction of the topic, discussion and completion of the documents, and periodic review and communication of directives. This stepwise design allows for thoughtful decisions and is less time consuming during an individual office visit. Consultation with family, friends, and clergy is also facilitated.

Types of advance directive instruments

The most common advance directives are the living will and the durable power of attorney for health care. A living will is a statement by a competent person expressing their desire for medical care in the event of future incompetence or inability to communicate. They may request or decline life sustaining treatments. Living wills are most often in the form of a written document. They may be general or specific. They are, however, unable to anticipate all possible clinical scenarios. Medical situations occur which living wills fail to address. In such circumstances, the durable power of attorney for health care is in-

valuable by assisting in the interpretation of the living will.

The durable power of attorney for health care (proxy) allows a competent patient to choose a spokesperson for health care decisions. "Durable" means that the document remains in effect after the patient becomes incompetent. This proxy is able to speak for the patient and their decisions have the same authority as those of the patient. Linking the durable power of attorney to the living will strengthens both instruments. The living will provides the patient's expressed views, while the durable power of attorney for health care expands those views in the event interpretation is necessary.

The process of completing advance directives

Obtaining sample documents of the living will and durable power of attorney for health care is important. The American Medical Association, American Bar Association, and American Association of Retired Persons have joined efforts in producing the brochure, *Health Care Advance Directives*.⁵ It introduces the principles and terminology of these instruments and serves as an adjunct to discussion. The Maryland Attorney General's Office will provide single copies of the living will and durable power of attorney (410-576-6300). These forms may be photocopied for patients. Concern for Dying, Inc. will furnish state-specific forms at nominal cost for those living in other states.⁶ Having this literature easily accessible is a critical step toward improving compliance.

The process of establishing advance directives is often physician-initiated. To begin discussion with the patient, use open ended statements. These are intended to be non-threatening. "I want to talk to you about planning for future health care. Many now recommend that people make plans to direct their care in case of future illness, and that doctors discuss these issues

routinely with their patients... It is part of helping to ensure that you are cared for the way that you would want to be. . . ."⁷ A brief description of the concept of the advance directive should then be presented. It is beneficial to have a nurse or other office personnel trained to assist. Patient instructional literature provides additional background and aids in both the effectiveness and efficiency of the process. Review of the literature should help patients formulate attitudes toward various types of life support. Discussion with family, friends, and clergy is encouraged as it helps clarify ideas and build consensus.

During the next visit, discussion should focus on conveying or clarifying medical information about life-sustaining treatments. The use of hypothetical scenarios is helpful. These scenarios should describe a range of disability and how that disability relates to the chance of recovery. Cases might include irreversible coma, progressive dementia, and chronic ventilatory support. During this discussion, the patient may describe certain clinical conditions as "worse than death." Attempt to define points at which patient attitude changes from desiring to declining treatment. The goal is to convert abstract values, spiritual beliefs, and life's experience into more tangible thoughts.

Once the patient is able to reasonably articulate their desires, the living will may be completed. The durable power of attorney for health care should be identified, and if desired, included in the office visit. Most states, including Maryland, do not require that legal counsel be involved in this process, although some request the documents be notarized.

Review and communication

The annual physical examination presents an opportunity for review of established advance directives. Include this as a part of preventive care. Significant changes in medical condition should also trigger reevaluation. Copies of these

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documents should be provided to those deemed appropriate, including family, clergy, and attorney.

The final critical step in the use of advance directives is communication of this information when needed. This can be challenging as patients move between health facilities. However, lack of such direction may result in care which is not desired.

Summary

Advance directives are a valuable form of medical planning and well-accepted by patients. They are part of preventive health care and should be included as routine care. Completion of these documents is a process requiring patient/physician communication and can be effectively completed over several visits. Periodic reassessment of patient attitudes is important. Finally, including these documents in the patient record and communication of this information is important, as it helps shepherd the patient through the medical system. Physician investment in the use of advance directives is minimal, while the rewards are prodigious.

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Myrrh: A forgotten analgesic compound

Gold, frankincense, and myrrh usually recall to Christians happy memories of the story of the birth of Christ and the three Magi from the East who brought these as gifts to the newly-born child.

Myrrh, a red gum, is isolated from *Balsamodendron*, a tree that grows in southwestern Arabia, Oman, and parts of Somalia. It was early recognized to possess some analgesic property.¹ The reason for its analgesic efficacy was never ascertained and the medical use of the compound was abandoned when opiates were discovered.

Interestingly, about two thousand years after its Biblical mention, nine Italian pharmacologists reported the isolation of three sesquiterpenes (hydrocarbons with the general formula of C₁₅H₂₂), two of which had analgesic effects blocked by naloxone, indicating an interaction with brain opioid mechanisms.² This activity could explain the use of myrrh as a pain killer in ancient times.

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Joseph M. Miller, M.D., a retired surgeon in Timonium, Maryland and Mary Ann Ayd, former managing editor, Maryland Medical Journal.

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- *New York Yankee Don Larsen threw the World Series' only no-hit game against Brooklyn Dodgers*

Cardiovascular surgery

Excerpted from the *Maryland State Medical Journal* 1956;5(7):379–380.

R Adams Cowley, M.D.

Today, heart disease is the leading cause of death in the United States. Compared with other diseases, heart disease takes a greater toll than the next five leading causes of death combined. In 1950, mortality statistics recorded that 745 000 people died from cardiovascular diseases. In second place, but far behind, stands cancer with 209 000 deaths; accidents 88 000 deaths; pneumonia 47 000 deaths; tuberculosis 34 000 deaths and in 6th place nephritis with 29 000 deaths.

Not only is heart disease the leading cause of death but it is a menace to all age groups. No age is respected. Let us look at this indiscriminatory potential killer more closely. At birth he may confront the newborn infant with any of the congenital malformations and, their consequences, if left untreated. Ascending the age scale to the 5 to 19 year bracket, rheumatic fever and its cardiac sequelae becomes the leading fatal disease in this group of young people. Beyond the age of 35 years cardiovascular disease outranks all other causes of death. After 45 years of age one out of every two deaths is caused by heart disease.

If we were to group these diseases, it would become apparent that congenital malformations account for only 2% of all heart disease while 90% of all heart disease can be attributed to rheumatic heart disease, coronary heart disease or hypertensive heart disease. Syphilitic heart disease is decreasing because of the use of penicillin. In an inverse ratio form, degenerative heart disease is increasing because the use of penicillin has prompted longevity. In the light of such information, it is difficult to discuss except very superficially, one field of cardiovascular surgery in the allotted time. For this reason, we shall only briefly touch on those conditions in which surgery plays a rehabilitative part and perhaps speculate about those conditions where surgery can play a greater part.

First, let us consider trauma to the heart partly because in this day of automobiles and speed, steering wheel crush injuries to the chest are common. The resulting contusions to the pericardial sac enclosing the heart and to the heart muscle are not unusual. Such pericardial or myocardial injury will produce either the symptoms of tamponade or infarction. If cardiac tamponade is suspected, aspiration is essential. Contusion is treated with 2 to 5 weeks of bed or chair rest.

A Look Back

Penetrating wounds of the heart are usually the result of stab and gunshot wounds. The presenting symptoms are those of cardiac tamponade: shock and often cyanosis. If unrecognized and untreated the end result is cardiac standstill. What is the treatment? Aspiration, transfusion and observation. If shock continues or recurs or aspiration fails, surgical intervention is the only treatment.

There is another type of cardiac tamponade of a chronic nature which is often forgotten — that of constrictive pericarditis. Its end result is as lethal as the acute type of tamponade. The treatment of this kind of cardiac tamponade is to surgically free the heart from its thick constricting pericardial shell.

There is no reason at this time to discuss congenital heart disease. All of us are aware of and grateful for the pioneer work

done by Dr. Blalock in pulmonic stenosis. With the proven results of shunting procedures and the newer intracardiac approach of Brock, surgery has become the choice of treatment. However, the importance of an accurate diagnosis cannot be overstressed. An error may subject a child to a needless and sometimes fatal operation. Two laboratory procedures which greatly reduce this error are cardiac catheterization and angiocardiology.

Septal defects, both atrial and interventricular, stand on less firm ground, as evidenced by the large number of surgical procedures devised and tried for an accurate closure.

In the field of acquired cardiac lesions, the most gratifying procedure is commissurotomy performed for patients with mitral stenosis. Every physician today is acquainted with the patient with mitral stenosis who after a mitral commissurotomy is now up and about, living an active and little restricted life. Even those less fortunate patients with degrees of mitral insufficiency are, on the whole, aided by cutting the valve commissures to produce a more nearly normal physiological valve leaflet.

Surgery for aortic stenosis has looked less convincing because of the high incidence of ventricular fibrillation at operation. With the newer technique of approaching the aortic valve from the aortic side this fatal complication has been eliminated and now the mortality and morbidity rates are rapidly decreasing.

The final field of cardiac surgery at present encompasses the coronary problem. Medically speaking and even with ideal care, all persons who develop coronary occlusion of one type or another are shown statistically to fall into one of four groups. 1) 25% will die of their first attack. 2) 25% will die within the first year. 3) 25% will die within a period of 5 years. 4) 25% will live 5 years or longer.

Medical treatment of coronary spasm and occlusion is purely supportive in nature. It would seem that the urgent need is a revascularization of the ischemic heart muscle by surgical methods. There are several procedures in order of magnitude to meet the risk, but all strive for one effect; to increase the blood supply to the impoverished myocardium.

Beck's work in this field has been monumental. His abrasive procedures with burr and talc are aimed at abolishing the trigger mechanism of localized myocardial ischemia. His arterial anastomoses to the coronary sinuses attempt to develop a greater cardiac vascular reserve. Vineberg with his mammary artery transplant and the other types of cardiopexies all strive to bring blood from some other source to the heart. It is hoped that selective vagal resection will relieve coronary vasospasm and improve collateral circulation. All of these procedures await the test of time but those which have been performed throughout the past few years boast better results than the classical medical regimes today. ■

Commentary

Reading Dr. Cowley's 40-year-old article on cardiovascular surgery instantly conveys the impact of the technologic revolution on medical care. During one surgeon's career, the complexion of his specialty changed entirely.

Forty years ago, rheumatic heart disease was a major part of a cardiothoracic surgeon's practice. Mitral commissurotomy was "the most gratifying procedure." Cardiac trauma, both blunt and penetrating, was treated by pericardial aspiration and rest. The Vineberg mammary artery transplant was a new idea, and the landmark shunt for blue babies, developed by Blalock, Taussig, and Thomas at The Johns Hopkins Hospital, was still novel.

Dr. Cowley mentioned cardiac catheterization and angiocardiology in his article. Three later technical developments revolutionized heart surgery in the second half of the century. The heart-lung machine changed cardiac operations from timid excursions on a dangerous medical frontier to routine procedures. Artificial heart valves cured congenital and acquired valvular disease. The electronic pacemaker, an early edition of which was invented by R Adams Cowley, cured conduction disturbances.

The pacemaker, prosthetic valves, and cardiopulmonary bypass, together with much more potent antibiotics and cardiac drugs, have made cardiovascular surgery one of the safest, most effective, and busiest surgical disciplines. R Adams Cowley, before he turned to the care of the accidentally injured, was one of its pioneers.

CHARLES E. WILES, III, M.D., FACS, FCCM

Dr. Wiles is medical school associate professor at University of Maryland Medical School and attending traumatologist and intensivist, R Adams Cowley Shock Trauma Center. ■

FROM THE BPQA

Editor's Note: With this issue of the *Maryland Medical Journal*, we are initiating a series from the Board of Physician Quality Assurance (BPQA). They will present actual cases heard by them, their adjudication, and the reasons for their decisions. It is hoped that this series will alert physicians to potential issues which could threaten their practice of medicine. Forewarned is forearmed.

■ Outpatient Detoxification of a Drug Abuser: The Case of Dr. A

The police reported the death of a 40-year-old man to the Board of Physician Quality Assurance (BPQA) because toxicology results indicated multiple drug overdose. One of the drugs in the deceased patient's system was darvon, which had been prescribed in large quantities by Dr. A. Dr. A had been seeing the patient for approximately six months prior to the patient's demise. During that six-month period, he saw the patient weekly and wrote prescriptions for darvon. Typically, 100 to 175 pills were prescribed on a weekly basis. Dr. A recognized that the patient was a substance abuser, and he was attempting to get the patient off this medication by gradually reducing the amount of medication he prescribed. When the patient overdosed, several other prescription drugs were also detected. Dr. A had not been prescribing these other drugs and was unaware that the patient was obtaining drugs from other sources.

■ Would you, in your capacity as a physician, prescribe a controlled substance to an individual who was drug dependent, but not taking the medication for a bona fide medical complaint? Is

it legitimate to prescribe in the manner described above, in an attempt to detoxify a drug-abusing patient? What would you do if you were a member of the licensing board and this case were presented to you?

Comment by Cheryl Winchell, M.D., Secretary/Treasurer, BPQA

Physicians who encounter drug-addicted patients have no obligation to provide prescriptions to prevent drug withdrawal. Arrangements should be made for an inpatient detoxification program. Federal law permits physicians to dispense medication for up to 3 consecutive days while a patient is waiting admission to such a program. Often when the day of admission arrives the patients fail to arrive as agreed. What if the patient says he can't afford an inpatient program? Should you then prescribe so that the patient won't go into withdrawal and die? If your answer is yes, you will fall into the same trap as Dr. A. Physicians providing treatment to addicted individuals must be registered by both state and federal authorities as an addiction treatment facility. So don't take the patient's problem and make it your problem by treating patients who decline to be treated in a registered facility.

A very different scenario applies when a patient has become drug-de-

pendent due to treatment for a legitimate illness. If the patient no longer needs the drug for medical reasons, the appropriate course is to gradually taper the drug over several weeks to prevent withdrawal symptoms. Generally, the patient will be anxious to get off the drug as soon as possible. In this situation, your goal and the patient's goal are the same and inpatient detoxification is rarely necessary.

One of the most difficult situations that physicians face is the treatment of patients who are former substance abusers who have a medical indication for narcotic pain relievers. In these cases, physicians need to draw on their experience with non-abusing patients to judge when it is time to begin to taper the patient off medication. Just as physicians have the obligation to treat patients' pain in an effective manner, so do they have an obligation to remove the addicting drug when the medical indications have been satisfied. Rather than have to face this difficult dilemma, many physicians will choose to under-treat a patient with a history of drug abuse rather than risk re-addiction. Truly, this is one of the situations where the art of medicine is put to the test.

■ Action taken by BPQA

The BPQA referred this case for peer review of the incident. The peer reviewers concluded that Dr. A had provided substandard care in his treatment of the patient. Although Dr. A's actions were well-intentioned, he showed poor clinical judgment in his evaluation, management, and treatment. Subsequently, the BPQA reprimanded Dr. A and ordered that he not attempt to detoxify patients who are already drug dependent through providing prescriptions of the addictive drug.

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*Some income may be subject to the federal alternative minimum tax. Income earned by non-Maryland residents will be subject to applicable state and local taxes.

**Morningstar proprietary ratings reflect historical risk-adjusted performance through 10/31/96. These ratings are subject to change every month. Ratings are calculated from the funds' 3- and, where applicable, 5-year average annual returns in excess of 90-day Treasury bill returns with appropriate fee adjustments and a risk factor that reflects fund performance below 90-day Treasury bill returns. The 1-year rating is calculated using the same methodology but is not a component of the overall rating. The Maryland Tax-Free Bond Fund received 4 stars for the 3- and 5-year periods. For the 1-year period, the fund received 5 stars and was rated among 1,728 municipal funds. The short-term fund received 5 stars for the 3-year period. For the 1-year period, the fund received 5 stars and was rated among 1,728 municipal funds. Ten percent of funds receive 5 stars, and the next 22.5% receive 4.

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Books, Etc.

Book review editor:
Chris Papadopoulos, M.D.

American Cardiology. The History of a Specialty and Its College.

W. Bruce Fye. Baltimore, MD: The Johns Hopkins University.
489 pages. \$24.95 (hardcover).

History teaches us where we come from, where we stand at present, and in what direction we are going. An excellent book on American cardiology was recently published; it is significant that the author is a clinician and a historian. Dr. W. Bruce Fye is chair of the cardiology department at Marshfield Clinic and a member of the faculty of medicine and of the history of medicine at the University of Wisconsin. He has made many contributions to medical history literature.

The present book is a history of cardiology as a medical specialty, in this country, in the early twentieth century and its evolution to one of the most significant fields in medicine. It also examines the development and impact of the American Heart Association and the American College of Cardiology.

Medical specialization developed gradually in the United States during the nineteenth century and expanded and became more pragmatic in the twentieth century as a result of the ever expanding knowledge from discovery, invention, and human experience. Furthermore, socioeconomic factors and market forces played a role. Increasing awareness of the prevalence of heart disease, technological innovations, government research funding, and the availability of health insurance contributed in the development of cardiology into a major academic and clinical discipline. The author emphasizes organizational dy-

namics rather than the specific scientific and clinical content of cardiology as a specialty. He focuses on the interactions and the complex roles played by individuals, organizations, academic institutions, and the federal government in the development of American cardiology. He looks closely at the creation and evolution of the American Heart Association and the American College of Cardiology and their territorial disputes, and also at the evolution of medical education for cardiologists. The impact on the future of medical specialization of present day issues, such as managed care and the government decreases in educational and research funding, are also discussed.

The author has produced an in-depth and comprehensive study of American cardiology. It is based on dozens of oral history interviews and extensive review of the literature and of many archival sources. The text is supplemented by important tables, figures, and historical photographs. A long list of references, historical notes, and a bibliography are provided.

This book is an important contribution that should be of interest to medical historians, cardiologists, other cardiovascular specialists, and anyone interested in the development of American medicine.

CHRIS PAPADOPOULOS, M.D., F.A.C.C.
Dr. Papadopoulos is chief of cardiology, emeritus, Harbor Hospital Center.



This is a CD-ROM collection of volumes 12 through 17 of the journal *Family Practice Recertification* (1990 through 1995). Most of the articles feature an audio introduction by the author and self-examination questions. The graphics for the figures and illustrations are superb, and the articles are thoroughly cross-indexed and easy to search. Pop-up screens are used for footnotes and references. Figures, illustrations, and text may be printed; however, the printout occasionally stops before the end of the article when printing the entire text.

The CD-ROM package includes an examination which can be completed and returned for 30 hours of category 1 CME. For those preparing for the family practice recertification examination,

this volume contains two issues that have questions adapted from the residents' in-training examinations, with answers and discussion. It is excellent practice, and it allows the reader to experience the types and style of questions on the exam. This practice, plus the \$10/credit hour CME, make it worthwhile for those who do not subscribe to the paper version of the journal, which at \$60/year is comparably priced.

Hardware requirements are a Windows or Macintosh system, 4 MB RAM, CD-ROM drive, sound card, and at least a 256-color display.

RICHARD MOORE, M.D.

Dr. Moore is Director of Public Health Service Health Unit #1 in Bluemont, Virginia. ■

NRHA announces debut of home page on the World Wide Web

The explosive use of the Internet as an information tool has brought a new dimension to the National Rural Health Association's (NRHA) efforts to connect health professionals in rural areas and to inform people of the unique problems facing health care in rural America. The NRHA's home page can be found on the World Wide Web at <http://www.NRHArural.org>.

Among other things, activities affecting rural health care, such as legislative initiatives, upcoming meetings and seminars, and new publications will be posted.

Headquartered in Kansas City, MO, with an office in Washington, DC, the NRHA is a nonprofit association composed of individual and organizational members who share a common interest in rural health. Its primary mission is to provide leadership for improving the health and health care of rural Americans through education, communications, research, and advocacy.

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205 (410-955-2959).

MGA\MGS series, Department of Mental Hygiene, The Johns Hopkins University, School of Hygiene and Public Health, Keswick Nursing Center, 40th Street, 6:00 -7:00 p.m., light refreshments served at 5:30 p.m. Credits: TBA. Sponsored by the Maryland Chapter of the American Geriatrics Society (AGS) and the Maryland Gerontological Association (MGA), with the assistance of the Geriatrics Committee of the Maryland Academy of Family Physicians. Info: Donna Meisel Weinreich, 410-675-3244 (e-mail: dmeisel@umabnet.ab.umd.edu) or Joseph J. Gallo, M.D., M.P.H., 410-955-0599 (e-mail: jgallo@welchlink.wlech.jhu.edu).

TB prevention in long-term care

Feb. 18

Perioperative management of the elder adult

Mar. 11

Pain management in the older adult

Apr. 29

Preventive geriatrics

Jun. 17

Frontiers in research and clinical management of asthma and allergy, Johns Hopkins Asthma and Allergy Center, 5501 Hopkins Bayview Circle. Credits: AMA Cat 1 credits, AAFP. Fee: \$395/physicians; \$275/residents, fellows, allied health professionals. Sponsors: the Division of Allergy and Immunology, the Division of Pulmonary and Critical Care Medicine, and the Asthma and Allergy Foundation of America, Maryland Chapter.

Jan. 17-19

Comprehensive endoscopic sinus surgery. Sponsor: Dept. of Otolaryngology, Head and Neck Surgery, Johns Hopkins Medical Institutions. **Registration is limited.**

Basic endoscopic sinus surgery. Hands-on laboratory program. Up to 9 AMA Cat 1 credits. Fee: \$895/ lab and lecture; \$350/ lecture only.

Jan. 23

Advanced endoscopic sinus surgery. Hands-on program for surgeon who has performed at least 25 cases. Up to 19 AMA Cat 1 credits. Fee: \$1495/ lab and lecture; \$550/ lecture only.

Jan. 24-25

Computed body tomography 1997: The cutting edge, sponsored by the Johns Hopkins Medical Institutions, Department of Radiology, at Peabody Orlando Hotel, Orlando, Florida. Comprehensive review of recent advances in computed body tomography with some correlation with MRI. Credits: 21 AMA Cat 1 credits, 21 Cat A CE credits as designated by the ASRT, CEUs from the Florida HRS Office of Radiation Control. Fee: \$575/physicians; \$500/residents, fellows, technologists. Contact: Conference Coordinator, JHMI, Office CME, 410-955-2959, Fax: 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

Feb. 6-9

14th annual gastroenterology update: multi disciplinary approach — an exercise in interactive gastroenterology, Silvertree Hotel, Snowmass Village/Aspen, Colorado. 19 Cat 1 AMA credits. Fee: \$495/physicians; \$375/residents, fellows, allied health professionals.

Feb. 9-14

Perioperative management: a course designed for practitioners to limit patient risk by proper pre- and postoperative evaluation and care, Marriott's Marco Island Resort and Golf Club, Marco Island, Florida. Credits: 20.5 AMA Cat 1 credits, 20 CE credits by the AANA. Fee: \$525/physicians (After Feb. 9, 1997: \$550); \$490/residents, fellows, and CRNs (After Feb. 9, 1997: \$515).

Mar. 9-12

Management of auditory and vestibular disorders and vestibular practicum. Credit: TBA. Fee: \$400/physician; \$300/residents, fellows, allied health professionals. Practicum: \$75.

Mar. 12-13

Second annual cardiovascular symposium with the experts, sponsored by the Department of Medicine at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 11.5 Cat 1 AMA credits. Fee: \$175 by March 30, \$225 after March 30. Contact: Conference Coordinator, CME office, JHMI, 410-955-2959, Fax: 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

Mar. 30-31

The Johns Hopkins Medical Institutions (continued)

- Diagnosis and treatment of neoplastic disorders.** Sponsor: The Johns Hopkins Oncology Center. Credits: 14 AMA Cat 1. Fee: \$300/Advanced registration (before Feb. 1); \$325 (Postmark Feb. 1 and after); \$150/residents, fellows, allied health professionals. **Apr. 3–4**
- Update on Alzheimer's disease and other dementias,** Renaissance Harborplace Hotel, Baltimore, MD. Up to 7 Cat 1 AMA credits. Fee: \$145/physicians; \$110/psychologists in practice; \$90/residents, fellows, allied health professionals. **Apr. 12**
- 25th annual pediatric trends,** Johns Hopkins Medical Institute, Department of Pediatrics. This course provides a comprehensive update on new developments of interest to practitioners who care for infants, children, and adolescents. Credits: 42.5 Cat 1 AMA credits, 45.5 AAP credits, 37.5 AAFP prescribed hours. Info: Program Coordinator, Office of Continuing Education, 410-955-2959, Fax: 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu). **Apr. 14–19**
- 38th annual postgraduate institute for pathologists in clinical cytopathology.** Sponsor: The Johns Hopkins University School of Medicine. Credits: 94.5 AMA Cat 1 plus up to 10 hrs. video instruction. Fee: \$2450/physicians; \$1300/senior residents.
- Course A** (Home study) **Mar.–Apr.**
- Course B,** concentrated lecture and laboratory studies, Johns Hopkins Medical Institutions, Baltimore, MD. **Apr. 14–25**
- 11th annual mood disorders symposium,** sponsored by The Johns Hopkins Affective Disorders Clinic, and DRADA. Credit: AMA Cat 1 credit, Cat A credit, Md. State Board of Examiners of Psychologists, Md. State Board of Examiners for Social Workers. Fee: \$50/DRADA members, \$60/other attendees. Info: Program Coordinator, JHMI, Office of CME at 410-955-2959, Fax: 410-955-0807. **Apr. 30**

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. 40 Cat 1 AMA/PRA credits. \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. 40 Cat 1 AMA/PRA credits. \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956) or by calling the phone number listed after a specific program. FAX 410-706-3103.

- Diagnostic and therapeutic advances in glaucoma management,** Sheraton Inner Harbor, Baltimore, MD. Credits: TBA. Info: Sharon Stenhouse, 410-706-3956. **Feb. 21**

University of Maryland School of Medicine (continued)

Self-Directed CME Activities

CD-ROM based interactive multimedia radiology teaching file for Mac or PC w/single-user licenses (SUL), site licenses (SL), or multisite licenses (MSL). 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available. Info: 410-706-3956.

Miscellaneous

Office of Continuing Medical Education, Washington University School of Medicine, Campus Box 8063, 660 South Euclid Ave., St. Louis, MO 63110-1093. Unless otherwise noted, seminars will be held at the Washington University Medical Center, Eric P. Newman Education Center (EPNEC), 320 S. Euclid Ave., St. Louis, MO 63110. Info: Cathy Sweeney, 1-800-325-9862, Fax: 1-314-362-1087.

Internal medicine review, Monday evenings, The Jewish Hospital.

Mar.–May

Integrated care of the thoracic surgery patient: a seminar for allied health professionals.

Mar. 21–22

Clinical pulmonary update.

Apr. 4–5

Refresher course & update in general surgery. The Ritz-Carlton Hotel, St. Louis, MO.

Apr. 10–12

Delmarva Foundation for Medical Care, Easton, MD. 3 Cat 1 AMA credits. Info: Roxanne Rodgers, 410-822-0697.

Health care improvement for physicians

Jan. 17

Health care improvement for physicians

Jan. 18

Health care improvement for physicians

Jan. 29

Health care improvement for physicians

Feb. 5

Health care improvement for physicians

Feb. 12

Health care improvement for physicians

Feb. 14

Health care improvement for physicians

Feb. 15

Health care improvement for physicians

Mar. 14

Health care improvement for physicians

Mar. 15

Medical care of women in the era of HIV disease (three-part series), The Medical and Chirurgical Faculty of Maryland, 1211 Cathedral St., Baltimore, MD, 7:00 -9:00 a.m. Credits: Maximum of 6 Cat 1 AMA credits available. Fee: \$15/session; \$40/series.

Successful patient education and counseling strategies for the busy practitioner

Jan. 15

Clinical management of the HIV-positive woman

Feb. 5

Clinical innovations in OB/GYN ultrasound, Renaissance Waverly Hotel, Atlanta, GA. Sponsors: Foundation for Health Education and Medical Education Collaborative. 15 Cat 1 AMA credits. Fee: \$595/physicians; \$475/residents, sonographers, allied health professionals. Info: 555 Route 1 South, Iselin, NJ 08830, 800-599-8878.

Jan. 17–18

Miscellaneous (continued)

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|--|-----------------------|
| Essentials of prostate & genitourinary imaging , Marriott's Orlando World Center Resort, Orlando, FL. Sponsors: Foundation for Health Education and Medical Education Collaborative. 13 Cat 1 AMA credits. Fee: \$595/physicians; \$475/residents, sonographers, allied health professionals. Info: 555 Route 1 South, Iselin, NJ 08830, 800-599-8878. | Jan. 17-19 |
| The modern medical and surgical treatment of cardiac heart failure , The Baltimore Museum of Art, Baltimore, MD. Sponsored by Helix Healthcare at the Union Memorial Hospital. Info: Laurie Russell, 410-554-6523. | Jan. 22 |
| Cardiovascular conference at Snowshoe , Mountain Lodge Conference Center, Snowshoe, WV. Sponsor: American College of Cardiology (ACC), 14 Cat 1 AMA credits. Info: Registration Secretary, Extramural Programs Dept, ACC, 9111 Old Georgetown Rd., Bethesda, MD 20814-1699. Info: 800-253-4636 ext. 695; Fax: 301-897-9745. | Feb. 3-5 |
| Magnetic resonance imaging of the brain, spine and musculoskeletal system , sponsored by the University of California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, CA. Credits: 30 Cat 1 AMA credits. Fee: \$695/physicians; \$495/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com.). | Mar. 2-7 |
| 4th annual update in general diagnostic imaging: breast, abdominal and neuroradiology imaging , sponsored by the University of Chicago, Department of Radiology, at The Breakers Resort Hotel, Palm Beach, Florida. Credits: 25 Cat 1 AMA credits (includes 15 hrs. in mammography). Fee: \$700/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail webmaster@ryalsmeet.com). | Mar. 10-14 |
| Minimally invasive therapy of the brain , sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Hotel, Marina Del Rey, California. Credits: 17.75 Cat 1 AMA credits. Fee: \$395/physicians; \$300/residents, fellows, and technologist. Info: Ryals & Associate, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Mar. 14-16 |
| Eastern wisdom and the practice of psychotherapy , The Conference Center at Sheppard Pratt, Baltimore, MD. Info: Barbara Johnson, Professional Education Programs, Sheppard Pratt Health System, 410-938-4598 (e-mail: riamy@capcon.net). | Mar. 22 |
| 1997 annual session, American College of Physicians , at the Philadelphia, PA convention center. The largest meeting for internal medicine and its subspecialties. CME credit available. Pre-session courses: March 20-21. | Mar. 22-25 |
| Problem solving in diagnostic radiology , sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Palm Beach, Florida. Credits: 30 Cat 1 AMA credits. Fee: \$694/physicians; \$400/residents, fellows, and technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Mar. 29 |
| 17th annual residents' radiology review course , sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, CA. Designed for senior radiology residents and practicing radiologists. Course covers all major modalities. Credits: 41 Cat 1 AMA credits. Fee: \$695/physicians; \$450/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-8959 (e-mail: webmaster@ryalsmeet.com). | Mar. 30-Apr. 4 |
| Reimbursement and managed care: essential reimbursement strategies in emergency medicine , Hyatt Regency, Baltimore, MD. Sponsor: the American College of Emergency Physicians (ACEP). 15 Cat 1 AMA credits, 15 Cat 1 ACEP credits. Info: 800-798-1822. | Apr. 3-4 |

Miscellaneous (continued)

- Breast imaging and interventions**, sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, CA. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$250/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 4–6**
- Getting control: effective procedure coding for emergency medicine**, Hyatt Regency, Baltimore, MD. Sponsor: the American College of Emergency Physicians (ACEP). 15.5 Cat 1 AMA credits, 15.5 Cat 1 ACEP credits. Info: 800-798-1822. **Apr. 4–6**
- Leadership conference**. Sponsor: the American College of Emergency Physicians (ACEP), Stouffer Mayflower Hotel, Washington, DC. CME credits: TBA. Info: 800-798-1822. **Apr. 6–7**
- Legislative issues forum**. Sponsor: the American College of Emergency Physicians (ACEP), Stouffer Mayflower Hotel, Washington, DC. CME credits: TBA. Info: 800-798-1822. **Apr. 7–9**
- Building a multidiscipline team for the diagnosis and management of breast disease**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Plaza Hotel, New York, NY. Credits: 21 Cat.1 AMA credits. Fee: \$595/hospital team (no fee for every fifth member — must register together to qualify). Info: Ryals & Associates, 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 10–13**
- Infectious disease '97: A comprehensive review for the practicing physician**, sponsored by The Center for Bio-Medical Communication, Inc. (CBC), at Renaissance Washington, D.C. Hotel (reduced hotel rates for course registrants). Credits: 18.25 Cat 1 AMA credits, 18.25 AAFP credits. Fee (on or before Feb. 3, 1997): \$495/physicians; \$350/physicians-in-training and other allied health professionals. Info: 201-385-8080, Fax: 201-385-5650 (e-mail: webmaster@ryalsmeet.com). **Apr. 11–13**
- Sixth annual meeting and clinical congress of the American Association of Clinical Endocrinologists (AAACE)**, Marriott, Philadelphia, PA. Up to 36.5 Cat 1 AMA credits. Info: 904-353-7878. **Apr. 16–20**
- Problem solving in imaging of the brain, spine, and head and neck**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Amelia Island, FL. There will be both didactic lectures and workshops. Credits: 26 Cat 1 AMA credits. Fee: \$650/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 17–20**
- 2nd annual angio/interventional review course**, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, FL. Credits: 10 Cat 1 AMA credits. Fee: \$215/physicians; \$155/residents, fellows, full-time military, U of F radiology alumni (\$135 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 19–20**
- 9th annual radiology review course: "What you need to know,"** sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, FL. Attendees will improve their knowledge of differential diagnosis, imaging patterns, and techniques of examination. Credits: 50 Cat 1 AMA credits. Fee: \$695/physicians; \$525/residents, fellows, full-time military, U or F radiology alumni, (\$475 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax: 701-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 20–25**
- Critical Care Medicine '97: 11th annual review and update**, sponsored by the Center for Bio-Medical Communication, Inc., Hyatt Regency, Washington, D.C. Credits: 41.25 Cat

Miscellaneous (continued)

1 AMA credits, 41.25 AAFP. Fee (on or before Mar. 21, 1997): \$795/physicians; \$575/physicians-in-training and allied professionals. Info: 201-385-8080, Fax: 201-385-5640 (e-mail: cbcbiomed@aol.com.).

2nd annual mammography review course, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, FL. Course is designed as an overview of the practical aspects of breast imaging, including interventional procedures. Credits: 15 Cat 1 AMA credits. Fee: \$295/physicians; \$215/residents, fellows, full-time military, U or F Radiology Alumni (\$185 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax: 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 25-27**

Self-Directed CME Activities

Maryland physicians' campaign against family violence, module one: domestic violence, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Fee: \$10.4 Cat 1 AMA credits. Info: 410-539-0872. Expires September 5, 1997.

Maryland physicians' campaign against family violence, module two: child maltreatment, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Fee: \$10.4 Cat 1 AMA credits. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

Fluorescein angiography conference, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00-9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.

Sinai Hospital of Baltimore medical grand rounds, Zamoiski Auditorium, Thursdays, 9:00 - 10:00 a.m. Info: 410-578-5528.



PHYSICIAN'S RECOGNITION AWARD

During October 1996, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

Frank Sidney Billingsley
Hollis Roberta Chaney
Theodore Edward Evans
Donald E. Garland
Duane Michael Gels

Stephen Alan Goldman
Uchechi T. Opaigbeogu
Charles R. Privitera
Howard Neil Robinson
William Russell

Elizabeth Orr Segal
Douglas Alan Smith
Mohammad Far Taleghani
Robert Joel Wilensky

Parris N. Glendening - Governor of Maryland

Martin P. Wasserman, M.D., J.D., Secretary
Department of Health & Mental Hygiene



J. Mehsen Joseph, Ph.D., Director
Community Health Surveillance & Labs Admin

Ebenezer Israel, M.D., M.P.H., Director
Epidemiology & Disease Control Program

EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

January, 1997

Scabies Outbreaks in Maryland on the Rise Epidemiology, Diagnosis, Treatment, and Prevention

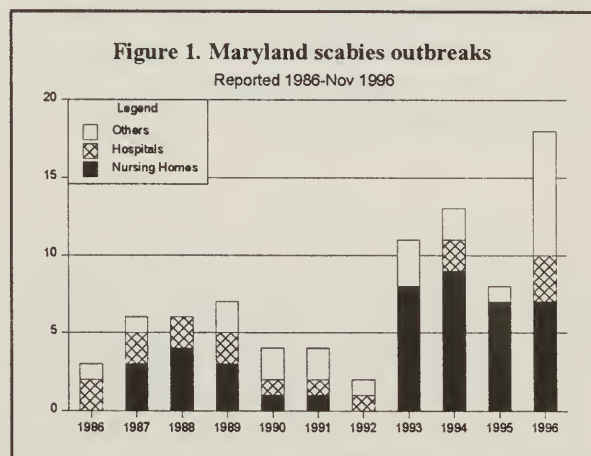
Maryland has seen an increase in the number of reported scabies outbreaks since 1993. In 1996 through November 15, 18 scabies outbreaks have been reported; 14 since September 1. The recent wave of scabies infestation in the USA and in Europe has evolved in the absence of major social disturbances and has affected people of all economic levels, without regard to age, gender, race, or standards of personal hygiene.

Epidemiology

Humans are the source of mite infestation, and transmission occurs most often by close personal contact since mites do not survive for more than 3 to 4 days without skin contact. Transmission occurs as long as a person remains infected and untreated, including the interval before symptoms develop. The incubation period in persons without previous exposure is usually 4 to 6 weeks, and those previously infected develop symptoms 1 to 4 days after repeat exposure.

In Maryland, scabies outbreaks have been reported mostly by nursing homes (52% of 82 reported cases from 1986 to present). Hospitals reported 20%, and other facilities such as schools, institutions, and clinics reported the remaining 28% of outbreaks. The average attack rate in nursing homes was 6

cases per 100 residents. No predilection for time of year or season is apparent. Figure 1 summarizes data collected by the Maryland Department of Health and Mental Hygiene (DHMH) from 1986 to present on scabies outbreaks by type of facility and year.



Clinical Manifestations

Disease signs and symptoms include an intensely pruritic, erythematous, papular rash. Itching is more intense at night. In older children and adults, frequently affected areas are the interdigital folds, flexor aspects of the wrists, extensor surfaces of the elbows, anterior axillary folds, belt line, thighs, navel, penis, areolae, abdomen, intergluteal folds, and buttocks. In children less than two years old

the rash is often vesicular, and is frequently found on the head, neck, palms, and soles. Excoriations are common. The lesions can be easily mistaken for other common rashes. A high index of suspicion must be kept when examining patients complaining of rash; especially when they are immunodeficient or from nursing homes. Occasionally, 2-to 5-mm red-brown nodules form on covered parts of the body such as the genitalia, groin, and axilla. These are scabies nodules, a granulomatous response to dead mite parts and feces, and can remain for weeks to months despite effective treatment.

A severe form of the disease, called Norwegian (crusted) scabies is seen in immunodeficient or senile patients. It appears as a generalized dermatitis distributed beyond where mites have burrowed, with extensive scaling and at times vesiculation and crusting. The usual severe itching may be reduced or absent. It is much more contagious than regular scabies because affected individuals are much more heavily infested: 2,000,000 mites per case of Norwegian scabies compared to 50 mites per case of regular scabies.

Diagnosis

Definitive diagnosis of scabies can only be made by microscopic identification of scabies mites, eggs, or scybala (feces) obtained from skin scrapings of infested patients. The scrapings must be taken from intact burrows or papules. Characteristic mite burrows appear as grey or white, tortuous, threadlike lines. Most burrows are obliterated by scratching long before a patient is examined by a physician. A good method to locate areas to obtain scrapings is to apply methylene blue dye to suspected skin sites, which outlines intact burrows. Then, sample collection is best done by applying mineral oil, microscope immersion oil, or water, and scraping the entire length of the burrow with a #15 scalpel blade.

Treatment

Adequate treatment requires applying a scabicide cream or lotion over the entire body, and changing to fresh clothing and bedding. Treatment of choice is one application of 5% permethrin (Elimite[®]). Permethrin should be removed by bathing after 8 to 14 hours. Alternatives are lindane (Kwell[®], Scabene[®]) and crotamiton (Eurax[®]). Itching may persist for 1 to 2 weeks after initial treatment, but should not be construed as treatment failure. This is an allergic reaction to remaining mite proteins and can be treated with antipruritic medications. For resistant cases the experimental drug ivermectin (Ivomec[®]) has been used with great success. It is given in a single oral dose of 200 mcg per kg (10-15 mg in adults), but to obtain it the FDA experimental drug request form (available from the FDA) must be completed.

Control Measures

- Treat prophylactically household members, healthcare providers, or others who have had skin-to-skin contact with an infested person.
- Ensure bedding and clothing worn next to the skin is laundered in a washer with hot water and a hot drying cycle. If clothing cannot be laundered it should be removed from the patient and stored for several days to a week or more to avoid reinfestation.
- After completion of treatment, children should be allowed to return to child care or school.
- Individuals with Norwegian scabies should be isolated and they and their close contacts must be treated promptly and aggressively to avoid outbreaks.
- Epidemics and localized outbreaks require stringent and consistent measures to treat contacts. Guidelines have been published and are available from the Epidemiology and Disease Control Program, DHMH (410-767-6677).

Standard Precautions

Summary of the New HICPAC Guideline for Isolation Precautions in Hospitals

In January 1996, the Hospital Infection Control Practices Advisory Committee (HICPAC) introduced their new Guideline for Isolation Precautions in Hospitals (*Infection Control and Hospital Epidemiology* 1996;17(1):53-80). Several questions may arise regarding the impact this guideline will have on the infection control practices used at health care facilities. The following list of anticipated questions and answers is presented in an effort to clarify the role of the HICPAC guideline.

Q. What are Standard Precautions?

A. "Standard Precautions" combine blood and body fluid precautions applied universally (i.e., "Universal Precautions") and body substance isolation and applies them to **all** patients.

Q. Why use Standard Precautions over Universal Precautions?

A. As Figure 1. illustrates, Universal Precautions protect against bloodborne pathogens only. Standard Precautions cover bloodborne pathogens, but also protect against pathogens spread by all other body fluids.

Q. What are transmission-based precautions?

A. The transmission-based precautions consist of additional measures designed to be used with Standard Precautions to further reduce

the risk of disease transmission. (Figure 2).

Q. How do I decide which transmission-based precautions to follow?

A. The HICPAC guidelines include a table of diseases and their respective transmission-based precaution category. Examples are given in Figure 2.

Q. Are there diseases that require more than Standard Precautions and transmission based precautions?

A. YES. Additional guidelines for tuberculosis can be found in the CDC Guidelines for the prevention of tuberculosis in health care facilities. MMWR 1994;43(RR-13):1-132.

Q. Do I still follow DHMH guidelines for specific diseases if I work in a long term care facility?

A. YES. The DHMH guidelines are consistent with the current HICPAC recommendations.

Q. What are the key points to remember about the new HICPAC guideline?

A. Figure 2 summarizes all of the key points outlined in the new HICPAC guideline.

If you have additional questions, please contact your local health department or the Maryland Department of Health and Mental Hygiene, Epidemiology and Disease Control Program at (410) 767-6677.

Figure 1. Standard Precautions Protect Against More Pathogens

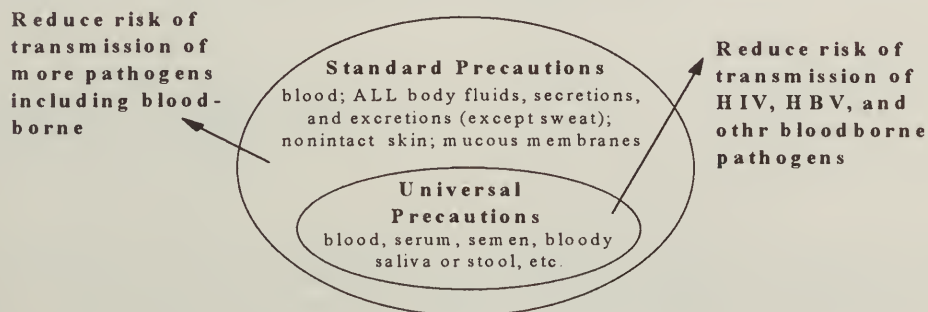


Figure 2. Summary of the Guidelines for Isolation Precautions in Hospitals: HICPAC Recommendations for Isolation Precautions in Hospitals (*Infection Control and Hospital Epidemiology* 1996,17(1):53-80)

Standard Precautions Summary

Standard Precautions are designed to incorporate the protection against blood-borne pathogens achieved by **Universal Precautions**, and the protection against other pathogens achieved by **Body Substance Isolation**. Standard Precautions are to be used on **ALL** hospital patients, regardless of their diagnosis or presumed infectious status, when coming into contact (or risk of contact) with any of the following: (1) **blood**, (2) **all body fluids, secretions and excretions except sweat**, (3) **nonintact skin**, or (4) **mucous membranes**.

Standard Precautions consist of the following nine components:

- (1) Routine hand washing
- (2) Consistent and correct glove use (i.e., glove changes with hand washing between patients)
- (3) Appropriate use of masks, eye protection, and face shields
- (4) Appropriate use of gowns [when necessary]
- (5) Routine cleaning or disposal of patient-care equipment
- (6) Regular cleaning of all environmental surfaces
- (7) Appropriate handling of contaminated linen
- (8) Strict adherence to occupational safety requirements
- (9) Effective management of patients with poor hygienic behaviors



Transmission-based Precautions Summary

Transmission-based precautions consist of **additional** measures designed to be used in addition to Standard Precautions to further reduce the risk of disease transmission. Transmission-based precautions are divided into the three categories listed below. Specific use of a category of transmission-based precautions is based upon the disease(s) of the patient. A partial list of disease examples are listed below; **for a complete list of diseases please refer to *Infection Control and Hospital Epidemiology* 1996,17(1):53-80, Appendix A.**



Airborne Precautions

- (1) Place patient in a private room or cohort.
- (2) Use respiratory protection when appropriate.
- (3) Limit patient transport within the facility.
- (4) Use additional precautions with tuberculosis.*

Example Diseases:

TB-pulmonary or laryngeal
Measles
Chickenpox



Droplet Precautions

- (1) Place patient in a private room or cohort; when not possible, maximize distance between patients.
- (2) Wear mask when working closely with the patient.
- (3) Limit patient transport within the facility.

Example Diseases:

H. influenzae meningitis
N. meningitidis meningitis
S. pneumoniae pneumonia
Diphtheria
Pertussis
Influenza



Contact Precautions

- (1) Place patient in a private room or cohort; when not possible, consult the infection control practitioner (ICP).
- (2) Wear gloves upon entrance to room and at all times.
- (3) Wash hands with antimicrobial soap upon leaving the room taking care not to touch environmental surfaces.
- (4) Wear a gown when entering the room if contamination is possible.
- (5) Limit patient transport within the facility.
- (6) Dedicate the use of personal, noncritical medical equipment to a single patient.
- (7) Use additional precautions for preventing the spread of vancomycin resistance.**

Example Diseases:

MRSA/ VRE infection or colonization**, C. difficile with diarrhea, Shigellosis if diapered or incontinent, Scabies

*CDC. Guidelines for preventing the spread of tuberculosis in health care facilities. MMWR. 1994;43(RR-13):1-132.

**HICPAC. Recommendations for preventing the spread of vancomycin resistance. AJIC 1995;16:105-13.

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Doctors from the University of Iowa recently conducted a study of the use of antibiotics during the first 200 days of life. The authors found that the use of antibiotics was common and increased with age, with 8.7% of the subjects having used at least 1 antibiotic by age 50 days and 70.5% of the subjects having used at least 1 antibiotic by age 200 days. The illness that prompted antibiotic use most frequently was otitis media; four other infections were also noted. Amoxicillin was the most frequently used antibiotic in this study (*Arch Fam Med* 1996;5:523-526).

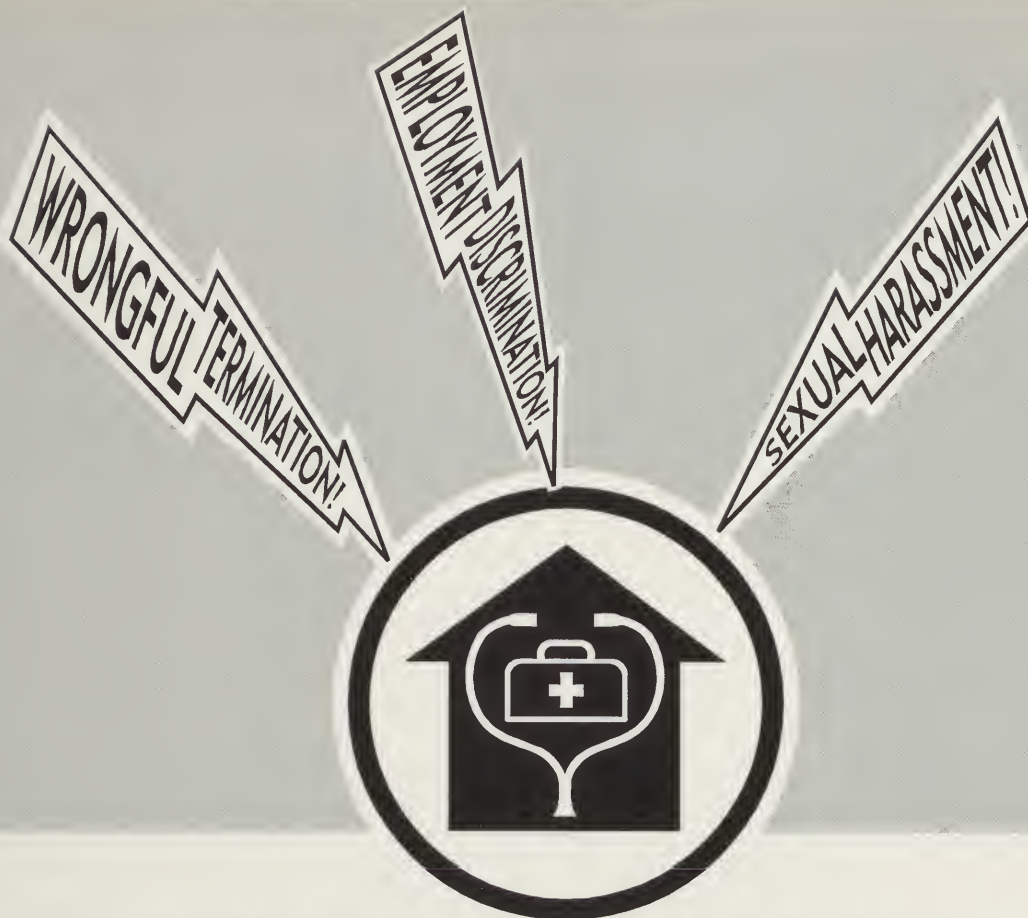
Using daily mortality data surrounding the January, 1994, Los Angeles County earthquake, doctors have established that earthquakes are a significant trigger of sudden cardiac death (*N Engl J Med* 1996;334:413). Reviewing deaths during the seven days prior to the earthquake, the day of the earthquake, and the six days after, they found a significant rise in the number of sudden cardiac deaths on the day of the earthquake, followed by a low incidence during the six days thereafter. The authors conclude that emotional stress can trigger sudden cardiac death in people who are vulnerable to such an event.

Hormone replacement therapy appears to increase bone density in amenorrheic runners. In a clinical study involving eight women runners with exercise-associated amenorrhea receiving hormone replacement therapy, and five women runners with exercise-associated amenorrhea not receiving therapy, bone density significantly increased in the therapy-receiving group. Nonsignificant bone density decreases were seen in the control group (*Arch Intern Med* 1996;156:2193-2195).

Three recently approved protease inhibitor class drugs, saquinavir (Invirase), zidovudine (Retrovir), and didanosine (Videx), are soon to be joined by a fourth, nelfinavir (Viracept). These protease inhibitors are the most potent antiretroviral agents available to treat patients with HIV disease. However, these protease inhibitors interact with rifampin derivatives, such as rifampin and rifabutin, which are used to treat and prevent the mycobacterial infections commonly observed in HIV-infected patients. A recent *MMWR Morbidity and Mortality Weekly Report* (1996;45:921-925) describes approaches and presents interim recommendations for managing patients who are candidates for, or who are undergoing, protease inhibitor therapy when tuberculosis is diagnosed.

Of hunters injured in incidents involving the intentional or unintentional discharge of the firearm of another hunter, approximately 73% were not wearing hunter orange clothing according to a study of New York hunters. Contributing factors for two-party hunting-associated firearm injuries recorded from 1989 to 1995 included being mistaken for game (35%), out of sight (22%), in line of fire (17%), unintentional discharge (11%), struck by ricochet (10%), and other/unknown (5%). For those injuries attributed to the highest contributing factor, being mistaken for game, 94% were not wearing hunter orange. Hunter education courses promote the use of hunter orange clothing, but it is only mandatory in 40 states; New York is not one of them. (*MMWR Morb Mortal Wkly Rep* 1996;45:884-887.)

A recent case-control study suggests that estrogen replacement therapy may be useful for preventing or delaying the onset of Alzheimer disease in postmenopausal women (*Arch Intern Med* 1996;156:2213-2217). Of the 3760 women who died during the 14-year study period (1981 to 1995), 248 were diagnosed with either Alzheimer disease or other dementia. Authors cite a major advantage of this study is that the women filled out their own health surveys, indicating medical history and use of estrogen replacement, many years prior to disease onset. Results indicated a reduced risk of Alzheimer disease and other dementias for estrogen users compared to nonusers. The lowest risk was observed for those who were long-term users taking high doses.



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Maryland Bond	★★★★	★★★★	★★★★	—
Maryland Short	★★★★★	★★★★★	—	—
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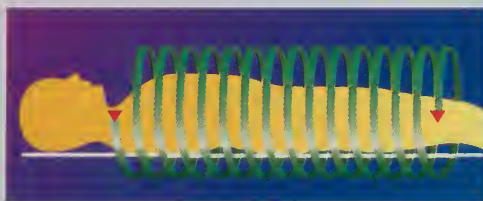
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INFORMATION FOR AUTHORS

For an information sheet on preparing manuscripts for submission to the editor of the *Maryland Medical Journal*, please call 410-539-0872 or 1-800-492-1056.

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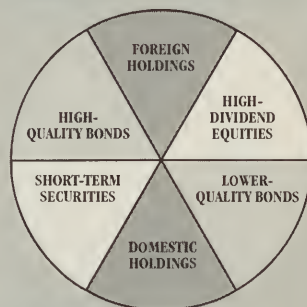
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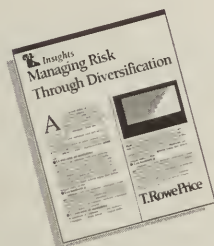
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The Scott-Key connection is interesting bit of Maryland medical history



The Scott-Key connection," published in the October, 1996, issue of the *Maryland Medical Journal*, is an interesting bit of Maryland medical history.¹ However, two slips of the pen occurred. Upton Scott was elected the first president of the Faculty in 1799 (not 1779) and he declined reelection in 1800 (not 1880).

Another bit of history about Francis Scott Key is his relationship to Roger B. Taney, a more-than-prominent native of Maryland. In 1806, Taney married Anne Phoebe Charlton Key, the sister of Francis Scott Key. After graduating from Dickinson College in 1795, Taney read law in Annapolis and began practice in 1799. He was a member of the Maryland House of Delegates and later was elected to the Senate. He was a leading lawyer in Maryland from 1815 to 1831. In the latter year, he was appointed Attorney General of the United States by President Jackson. In 1836, Taney was nominated and confirmed as Chief Justice of the United States to succeed John Marshall and had one of the longer tours of

office (1836 to 1864). Perhaps his best known decision was made in the Dred Scott case. The only point really decided there, however, was that Scott was a slave. Taney also declared that negroes had not been regarded as citizens by the framers of the Constitution and could not become citizens of the United States. He stated that the Missouri Compromise was unconstitutional and, therefore, Congress was bound to protect slavery in the territories.

JOSEPH M. MILLER, M.D.

Dr. Miller is a retired surgeon in Timonium, Maryland.

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1. Chase HV. The Scott-Key connection. *Md Med J* 1996;45:859-860.
2. Fleming WL. Taney, Roger Brooke. *The Encyclopedia Americana*. New York: Americana Corporation, 1954, volume 26, 240-241.
3. Chapelle SE, Baker JH, Esslinger D, Ridgway WH, Russo JB, Schulz CB and Stiverson GA. *Maryland. A History of Its People*. Baltimore: The Johns Hopkins University Press, 1986. ■

Slipped disk (ette) syndrome strikes

Author responds

The errors of the dates 1779 and 1880 in the published article were due partially to a slipped disk, the "slipped disk (ette) syndrome."¹ The 1779 date was a slip of my pen and the 1880, the disk's.

The article may be considered as a historical vignette to point out a connection between the 250th anniversary of the founding of Frederick County, Maryland and the bicentennial celebration of the Medical and Chirurgical Faculty of Maryland.

Francis Scott Key is scheduled to be used as the logo for the Frederick County celebration in 1998, and Dr. Upton Scott's history will surely be explored in preparation for the celebration in 1999.

HENRY V. CHASE, M.D.

Retired from the practice of internal medicine, Dr. Chase lives in Frederick, Maryland.

Reference

1. Altus MA. Medical Writing: Slipped disk(ette) Syndrome. *Md Med J* 1996;45:855. ■

Erratum

On page 1009 in the December 1996 issue, David Drewer's letter to the editor, paragraph three, should have read "...and in 1849, Moran found Poe 'in a violent delirium' which lasted four days, in 1885 the statement that 'Poe was four days in a fit of delirium' is 'utterly untrue.'"

Alan Nagel, M.D., is one of three authors of "Proximal Tibial Osteotomy," which appears in the September 1996 issue of *The Journal of Bone and Joint Surgery*. In this article the authors performed a retrospective study of the results of proximal tibial osteotomy and tried to determine the highest level of activity achieved by patients after surgery. Dr. Nagel has an orthopaedic surgery and sports medicine practice in Frederick.

Attila Nakeeb, M.D., Henry A. Pitt, M.D., Taylor A. Sohn, M.D., JoAnn Coleman, R.N., Ross A. Abrams, M.D., Steven Piantadosi, M.D., Ph.D., Ralph H. Hruban, M.D., Keith D. Lillemoe, M.D., Charles J. Yeo, M.D., and John L. Cameron, M.D., are among the authors of an article which appears in the October 1996 issue of *Annals of Surgery* (224: 63–475). The objective of the article, "Cholangiocarcinoma: A Spectrum of Intrahepatic, Perihilar, and Distal Tumors," is to introduce a simple method of classifying cholangiocarcinomas and to apply this system to analyze a large number of patients from a single institution. Ms. Coleman and Drs. Nakeeb, Pitt, Sohn, Lillemoe, Yeo, and Cameron are with the department of surgery; Drs. Abrams and Piantadosi are with the department of oncology; and Dr. Hruban is with the department of pathology, Johns Hopkins Medical Institutions.

Robin G. Chernoff, M.D., is one of three authors of "Attitudes of Academic Pediatricians With a Specific Interest in Child Abuse Toward the Spanking of Children," which appears in *Archives of Pediatrics & Adolescent Medicine* (1996;150:1049–1053). The authors conclude that most academic child abuse professionals believe that spanking is inappropriate and their beliefs are influenced by the context in which spanking occurs. Dr. Chernoff is with The Johns Hopkins University School of Medicine.

Ernest M. Moy, M.D., M.P.H., and Barbara A. Bartman, M.D., M.P.H., are co-authors of "Race and Hospital Discharge Against Medical Advice," which appears in the *Journal of the National Medical Association* (1996;88:658–660). The authors conclude that their results are "consistent with work showing that racial minorities are less satisfied with

the medical care they receive." Dr. Moy is with University of Maryland Department of Medicine. Dr. Bartman is with the Baltimore Veteran's Administration Medical Center, and is also an assistant professor of medicine and epidemiology at the University of Maryland School of Medicine.

Theresa Greene Reed, M.D., and C. Carnot Evans, Jr., M.D., are co-authors of "Essayists, essays, and hosts: Daniel Hale Williams Medical Reading Club," which appears in the *Journal of the National Medical Association* (1996;88:663–677). The article spotlights the 66-year-old independent reading club which meets six times a year for dinner and fellowship, and to consider topics relative to all specialty fields. The article gives a historical list of these meetings, naming essayists, topics, hosts, and the sites of the meetings.

Grover M. Hutchins, M.D., and Illy Dominitz, B.S., are two of the authors of "Focal Myocardial Ischemic Necroses Associated with Unstable Angina Pectoris," appearing in the *Journal of the American College of Cardiology* (1996;28:910–914). The authors' objective was to determine whether myocardial lesions develop in association with unstable angina pectoris. Dr. Hutchins and Mr. Dominitz are with the autopsy pathology division, department of pathology, The Johns Hopkins Medical Institutions.

Lisa A. Beck, M.D., Cristiana Stellato, M.D., Donald Leopold, M.D., Fuad Barody, M.D., and Bruce S. Bochner, M.D., L. Dawson Beall, M.S., and Robert P. Schleimer, Ph.D., are among the authors of "Allergens, IgE, Mediators, Inflammatory Mechanisms: Detection of the Chemokine RANTES and endothelial adhesion molecules in nasal polyps." Drs. Beck, Stellato, Bochner, and Schleimer are with the department of medicine, division of immunology, and Drs. Leopold and Barody are with the department of otolaryngology-head and neck surgery, Johns Hopkins University School of Medicine, Johns Hopkins Asthma and Allergy Center. Mr. Beall is associated with Otsuka America Pharmaceutical, Inc., Rockville.

Speak Out

Dead but not deserted.

Retrospective diagnosis: The potential and the pitfalls

As this past Halloween came around, Marylanders' thoughts once again turned to Edgar Allan Poe and "The Raven." During the last twelve months, over 15,000 people made the pilgrimage to Poe's grave, which continues to be a popular haunt of literary and spook enthusiasts alike. Well-known Italian stonemason Aldo Lagomarsino renovated Poe's burial monument in time for this past All Soul's Night. This was especially timely given the extra attention Poe's corpse has recently received.

The cause of Poe's demise has been reanalyzed. When the poet died in Baltimore in 1849, his death was attributed to congestion of the brain and cerebral inflammation. That account was challenged this year by Dr. R. Michael Benitez of the University of Maryland Medical System. Presented with a "blind" case history, Benitez swiftly recognized that his patient was not of this time and that the cause of death should be reexamined. He strongly raised the possibility that his patient (whom he later discovered was Poe) may have died of rabies.

Physicians and historians have good reason to applaud such an imaginative challenge to conventional wisdom. Both professional groups tend to seek answers and interpretations that more nearly approach human reality and experience as we understand them in our time. Physicians compile case histories through carefully constructed series of questions that are matched against conclusions derived from physical examinations. Those histories remain subject to change on the basis of further evidence, differing interpretations, and the evolving understandings of the disease. Historians begin with an idea, which in many ways is analogous to seeing a new patient. They too work backwards, asking questions and accumulating evidence until they think they are as close to the answer or correct interpretation as possible.

These tasks have recently become easier for both professions. Physicians now command a battalion of diagnostic procedures that were not available to earlier generations. These procedures produce a vast quantity of evidence that can be used to reach more exact conclusions. The limited and often vague disease classifications of previous centuries have given way to more precise diagnoses using modern scientific categories. Our specialized knowledge and technological sophistication can now be put to good use in reexamining case histories from the past, especially when a body still exists. DNA and radio-isotope testing are just two examples of techniques that can provide us with fresh evidence about the dead of years, and even centuries, gone by. Such new scientific information also helps historians as they assemble and reassemble the pieces in their historical jigsaw puzzles. In history, as in medicine, our access to information has increased remarkably since the dawn of the computer age. Internet and e-mail make communication between historians and libraries, just as between physician's offices and laboratories, routine even across great distances.

Speak Out

Shared information enhances our ability to create improved interpretations in both medicine and history.

Physicians and historians encounter similar problems in arriving at their conclusions. Usually, the further back in time the history or case history is, the skimpier the evidence will be. For the clinician there is less, or no, laboratory and radiographic information. For the historian there is less, or no, written, oral, or photographic record. In both instances, this means making more out of less; it means taking small fragments of information and trying to piece them together in a way that makes sense. Such a painstaking task raises the error level and sometimes raises as many questions as it answers.

To complicate matters in medicine even further, diseases are not static or standardized in their impact. Many of the bacteria and viruses responsible for infections mutate rapidly. Some that cause noteworthy diseases today are of relatively recent origin. Some long-standing diseases have varied virulence over time and place. In modern times, we are well aware of the difficulties attendant upon developing effective annual influenza vaccines when the viral strains shift and drift so readily. The same process is now frustrating scientists' efforts to produce a vaccine that will stand a chance of remaining effective against HIV. Certain other microorganisms that once resulted in extremely high mortality rates have come to elicit relatively benign responses in their hosts. Measles, which had a very low death rate in Western Europe, was a deadly illness when transmitted across the Atlantic by Columbus' crew to an immunologically naïve population. Likewise, smallpox resulted in far higher death rates when introduced to Native Americans than it did amongst European peoples who had over generations developed some degree of resistance.

Even greater interpretative problems cloud the historical lens when non-infectious etiologies are added to our list of variables. Public health conditions vary immensely both geographically and chronologically, and even within the same time period. In the state of Maryland 150 years ago, water supplies were quite different in Baltimore City, Frederick, and on the Eastern Shore. Population density, housing, ventilation, nutrition, proximity to malaria-infested swamps, and exposure to rats were just a few of the many factors that affected disease and health. These factors in turn could be substantially influenced by socio-economic status, race, gender, and age. When all these elements are taken into consideration, the task of reconstructing the past becomes extremely complex.

Thus, physicians, historians, and classicists have struggled for years to achieve a better understanding of Thucydides' account of *The Peloponnesian War*. This brilliant monograph provides a contemporary record of the fierce struggle between two quite different societies—the more civilized Athenians and the more primitively-organized Spartans. In the course of his narrative, Thucydides (c. 455 to 400 B.C.) discussed a terrible plague that he asserted “did more harm than almost any other single factor” (Book one, chapter one). The term “plague” has an array of meanings in different times and different places, and while many have speculated about the nature of the Peloponnesian plague, no consensus has emerged. Still, we continue to probe that problem and others in retrospective diagnoses, perhaps because much of the joy in this enterprise comes from the very process of speculation.

Speak Out

Thucydides' tome illustrates how we need to understand our sources intimately when we engage in retrospective diagnosis. Clearly, sources are crucial ingredients that shape the supply of evidence. Thucydides prided himself in being a careful student of his time and many Thucydidian scholars have commented on his almost scientific account. Yet, it is important to remember that the ancient historian was far from unbiased. He was an Athenian who fought in the war and was filled with a deep sense of tragedy at the loss of his beloved empire. Not only do we have to consider the biases of those who have produced historical materials, including case histories, but we have to be aware of our own when we raise questions about, and seek to reinterpret, the past.

These problems notwithstanding, retrospective diagnosis is an intriguing aspect of medical history and present day medicine. Dr. Benitez has done us all a favor in seeking to open doors that many assumed closed. He has encouraged us to rethink the answers to some old questions. Poe's case certainly contains mysterious elements. Despite the new analysis, I find it hard to ignore the fact that the poet had a history of alcohol and opiate abuse and that he was found in a semiconscious state stretched over two barrels outside Ryan's Saloon on Lombard Street. Perhaps his grim reaper wore the guise of rabies; perhaps not. We may never know, but we can be sure that Edgar Allan Poe would enjoy the added mystery and the speculation.

JANE ELIOT SEWELL, Ph.D.

Dr. Sewell is medical historian, the Medical and Chirurgical Faculty of Maryland. ■

Is Hippocrates dead yet?

I know that some of the original Hippocratean Oath is archaic, but are we physicians really ready to say that we'll abandon the oath of trust and confidentiality between doctor and patient? I know too that the amount of information circulating through managed care files and record rooms is impossible to fully protect, but are we willing to sanction a precedent-setting state statutory relinquishment of privacy by acquiescing to the non-consensual, patient-specific, government-administered Maryland Medical Care Data Base?

Many of you have heard the director of the Health Access and Cost Commission (HCACC), Mr. John Colmers, speak of his intent to protect the confidentiality of the vast numbers of citizens whose specific data are part of this data base. He is avuncular, reassuring, and credible sounding in his presentations to the various medical societies. However, he steadfastly and consistently avoids the same queries, time after time. I believe that is because he cannot give answers that physicians must hear on behalf of their patients.

He cannot reassure us that there will not be rogue employees willing to sell HCACC data tapes to unscrupulous purchasers. He cannot argue with the very real risk of using cross-referencing and probabilistic linkage electronic techniques to decode HCACC data tapes. He cannot refute that some zip codes have very few families living in them, making citizens in those areas especially susceptible to name identification. He knows that the

Speak Out

statutory right remains, to be exercised when HCACC chooses, to collect all data on all citizens, including self-pay patients, by having physicians directly provide these data without consent to HCACC. He cannot say why not allow consent or why not use aggregate data. He has agreed, when pressed (and in writing), that no security system is foolproof and HCACC cannot guarantee confidentiality.

Mr. Colmers cannot say why each of our patients must have his or her confidentiality jeopardized in order to set our fees as physicians — HCACC's bottom line goal. He only says "we need these data to separate fact from fiction" about health care expenditures. I find that to be a vague justification when the traditional standard requires compelling need—dangerousness—to bypass a patient's consent before information is divulged.

This September, a computer disc was delivered to two newspapers in Tampa, Florida. It contained medical data on 4,000 AIDS patients from the health department, data secured under lock and password with "only three people allowed access." These "safe and confidential" data clearly were neither. We're recklessly naïve if we think HCACC is immune to that.

There are two obvious solutions, either of which would salvage confidentiality. The first is to only gather aggregate, non-patient-specific data from the third party payors. These data could not be decoded and would have no worth on the burgeoning data market. Nothing in HCACC's first research report required patient-specific data.

The second solution is to ask patients' consent. The beauty of pressing for legislation that requires patient consent is that if patients believe in the data base and HCACC's research, they give their consent. If some people feel strongly about guarding their privacy, they withhold consent, as should be their right. If too few people give consent to establish statistical validity, then a plebiscite has been held, and as all current polls reflect, privacy will have been declared our patients' greatest priority.

Last May, HCACC received favorable press for "voluntarily" deferring gathering data on self-pay patients and for deleting a unique and specific patient identifier number and the exact day of birth. Now the Health Services Cost Review Commission (HSCRC) will be gathering those same deferred items, as well as other very specific patient identifiers, again without consent, from all hospital-based ambulatory care patients. These data tapes are accessible by HCACC and could easily be used to flesh out HCACC's data base, filling in for all advertised data deferments. That's called an end-run, and it is not HCACC's first (see *N Engl J Med*, 1996;335:889-900). Unless the enabling statute is changed in the legislature to require consent or the collection of only aggregate data, we can expect more of those; after all, this is a vivid case of the fox guarding the confidentiality chicken house.

Can we do anything about this? Absolutely. Find out where your legislators stand on this issue, and don't let them pretend this data base is impermeable. Pass out copies of the following notice to your patients so that they know the situation and can speak out too if they choose. We can fight to breathe life back into the venerable but moribund Hippocrates.

JENNIFER A. KATZE, M.D.

Dr. Katze is the chairperson, Maryland Psychiatric Society committee on privacy and a member of the American Psychiatric Association committee on confidentiality. ■

NOTICE TO ALL PATIENTS USING INSURANCE, MEDICARE, OR MEDICAID

As your physician, I think it's important to notify you that the state of Maryland enacted a law (HB 1359) in 1993 that is now having a direct impact on your right to privacy and to consent to the release of your medical information.

When physicians send information about you and your medical condition to your insurance company, they are permitted to do so only because you sign your consent. This is the way you are given an opportunity to know about consent to the fact that physicians are sending information about you to the insurance company. This is done for the sole purpose of your getting reimbursement for your health care.

However, the state of Maryland now intends to gather personal medical information about all health care received by most citizens. It is collecting data directly from insurance companies and Medicare/Medicaid, without notice to you, and without your permission. The data being collected in the state computer data bank includes an encrypted social security number or other number developed by your insurance company. Your medical treatment and diagnoses, sex, zip code, and date of birth are included without encoding. Over time, all your medical diagnoses and treatments will be accumulated. The state collects the data to do research on medical cost containment. Although physicians have always supported health care research, we strongly oppose governmental access to and electronic storage of medical information without patients' consent, because such private information belongs to each patient — not to anybody else, and because of the risk of confidentiality leaks.

The Health Care Access and Cost Commission is the branch of the Maryland state government that collects and studies the data collected. They believe they can protect the confidentiality of this information. Unfortunately, no computer security system is foolproof, and unauthorized leaks have occurred in other governmental data base systems. For that reason, the state data collection puts your medical privacy at risk. Since it is your private information, your permission should always be obtained before it is exposed to such risk.

Confidentiality is the foundation of the trust between patient and physician. That's why physicians place such a high priority on protecting it. In 1996 most physicians and many others concerned about privacy fought for new legislation that would have prevented medical information being collected by the state government without patients' consent. However, it was defeated. We will continue to advocate for laws that guarantee greater confidentiality.

If you have further questions about this matter, feel free to discuss this with me, or call the Maryland State Medical Society (Med Chi) at 410-539-0872 or 1-800-492-1056.

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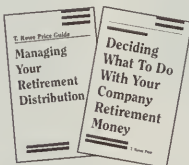
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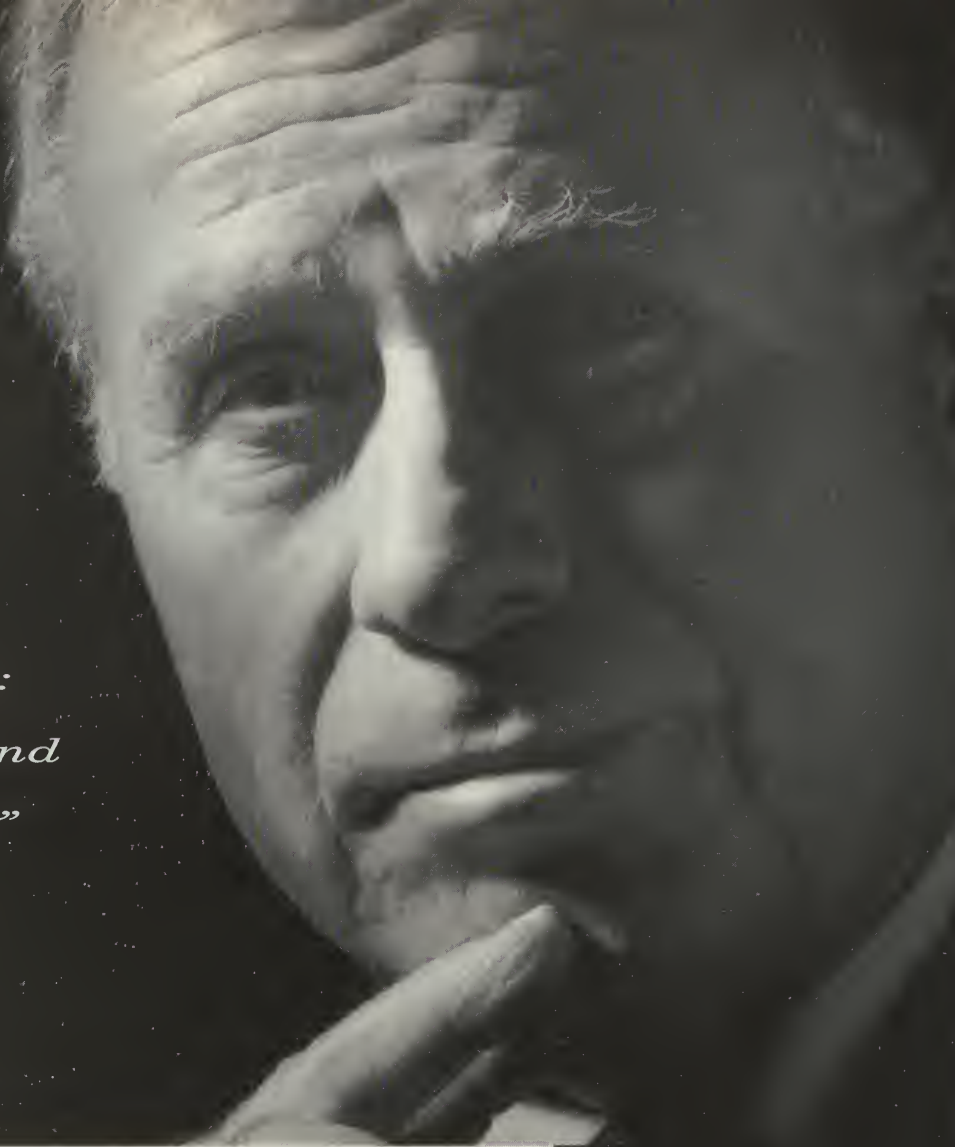


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Overview of implantable cardioverter-defibrillator in reducing total mortality in the high-risk coronary patient

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ABSTRACT: *The idea of an implantable cardioverter-defibrillator (ICD) was conceived in the mid- to late 1960s, and working circuits were built and tested at Sinai Hospital of Baltimore in the fall of 1969. After a number of years of pre-clinical testing, the device entered clinical trials at The Johns Hopkins Hospital in February, 1980, and received Food and Drug Administration (FDA) approval in 1985. The device appeared to be highly effective, but there was criticism that it had not been tested in a randomized fashion, and there was the feeling that drugs would eventually prove to be superior. In 1989, a series of randomized clinical trials were begun. One of these trials, the Multicenter Automatic Defibrillator Implantation Trial (MADIT), has concluded and the outcome has recently been published. The results are landmark in importance and lead the way towards rational treatment of serious ventricular arrhythmia patients in clinical practice.*

Disclaimer: *The author has been intimately connected with the development and commercialization of the implantable cardioverter-defibrillator (ICD) since its inception in the late 1960s. He was, successively, a consultant to Medtronic from 1970 to 1972, Medrad/Intec Systems, 1972 to 1985, CPI/Guidant, 1985 to 1989, and vice-president of medical sciences and senior consultant for CPI/Guidant, 1989 to 1994, and 1995, respectively.*

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The history of the implantable cardioverter-defibrillator (ICD) dates back to the mid-1960s. Even prior to 1969, the coronary care unit (CCU) experience showed that sudden cardiac death was a major public health problem, ventricular fibrillation was the mode of exodus, and prompt defibrillation of the heart was lifesaving.¹ On the other hand, many thought an effective drug would prove to be the solution, if indeed one did not already exist and the only problem was its proper use had not yet been defined.²

Dr. Michel Mirowski conceived the idea of linking the monitoring and defibrillation protection of the CCU on a relatively permanent basis to this problem, and came to Sinai Hospital of Baltimore as head of the coronary care unit to pursue this work.³

Working with him, we were able to achieve demonstration circuits within only a few months. They were battery-powered, automatically diagnosing and applying the treatment directly to the heart, recycling if needed, and using only commercially available components. Loss of the phasic nature of the right ventricular pressure curve was used as the monitoring signal.^{4,5} Defibrillatory shocks were applied through intravascular electrodes about 20 seconds after the onset of induced, otherwise lethal arrhythmias.⁶

Within a few years, we achieved fully-implantable defibrillators not much larger in size than the implantable pacemakers of the era. A major step forward was the filming of a number of episodes of automatic resuscitation by an implanted defibrillator in a conscious animal.⁷ In all, we accumulated chronic implant data spanning 60 months in 25 animals and had 24 episodes successfully reverted. In addition, the device underwent rigorous environmental and interference testing.⁸

We were at last ready for implantation in a human. The first patient underwent implantation by Dr. Levi Watkins, Jr. at The Johns Hopkins Hospital on February 4, 1980. The device was tested by inducing ventricular fibrillation and functioned similarly to the episodes in the experimental animal.⁹ Hopkins remained the only clinical implant center for a number of years, and then the study was slowly expanded to other institutions culminating in FDA market release in 1985.

During this time, because of the high apparent efficacy of the intervention, and the dismal outlook without it, there was little enthusiasm for enrolling patients into a randomized trial. The apparent success of the therapy had been great, quickly becoming regarded by most as the "gold standard" in the treatment of malignant arrhythmias.¹⁰⁻²² Although there was considerable circumstantial evidence of efficacy, in the later part of the 1980s, criticism started to be leveled that a proper randomized trial was never done, mortality might merely be shifted from one mode to another, and that the degree of efficacy was likely to be vastly overstated.²³⁻²⁷

Thus, since 1989, a number of randomized ICD trials were started in different clinical situations and substrates. Although most are still ongoing, one called the Multicenter Automatic Defibrillator Implantation Trial (MADIT) has terminated. Overwhelming superiority of the device limb with respect to total mortality was found by the data and safety monitoring committee on March 21, 1996, and the executive committee and the investigators concurred and officially terminated the study three days later.²⁸

This large-scale, prospective, randomized trial study, under the direction of The University of Rochester as the coordinating center, had 32 implanting centers, 30 in the United States, and 1 each in Germany and Italy. It began on December 27, 1990, and at termination, had randomized 196 high-risk coronary artery disease patients. There was an average follow-up of 32 months.

Patients considered for entry had transmural myocardial infarction (at least three weeks old), EF below 0.36, and 3-30 beat runs of nonsustained ventricular tachycardia (NSVT) on Holter recordings.²⁹ Previous work has shown that NSVT patients who are inducible at electrophysiologic study (EPS) and non-suppressible despite intravenous procainamide have a high subsequent mortality rate (some 30% at 2 years).³⁰

Thus, the patients were tested by EPS, and those positive and non-suppressible were randomized to ICD or conventional therapy. Baseline variables were extremely well-matched in the two treatment groups. There was no perioperative mortality, and the cross-over rate and the number of adverse reactions were low. The conventional limb patients had largely been put on amiodarone, generally acknowledged as the most effective currently available antiarrhythmic drug.

The study was monitored for early efficacy or possible harm using a highly efficient sequential design. At the termination of the trial, a total of 54 deaths had occurred, 39 in the conventional treatment mode and 15 in the device limb, a reduction of total mortality of 54% ($p=0.009$). The 95% confidence limits of the hazard ratio were from 0.26 to 0.82, far away from including the numeral one, which would have indicated equal outcome in both groups, and thus no benefit.

Additional findings of significance were that the number of transthoracic and transvenous leads (which had become available in the middle of the study) were evenly balanced, with similar outcome results for both groups. In a Cox regression model, there was no effect of antiarrhythmic medication, no changes when co-variables were adjusted for, and the hazard ratios were similar in all subgroups tested.

All told, the great significance of this trial is that it is the first large-scale randomized trial of an ICD, the first use of a sequential design in a device trial, and the first test of a prophylactic device use strategy.

The initial reaction from some colleagues was the general sentiment that "the typical MADIT patient is rare indeed." The implication was that these results, therefore, have little or no consequence for most clinical practice.³¹ However, this

is not true. A recent on-going large-scale multicenter trial indicates that the incidence of inducibility of coronary artery disease patients with EF of 0.40 or less and asymptomatic NSVT is 30%.³²

So the real question is one of finding efficacious treatment. Antiarrhythmic drugs, and specifically amiodarone, despite potent anti-arrhythmic powers, have been associated with sudden death rates of about 10% at one year and over 20% by 5 years.³³

Over the 16 years since the ICD was introduced into clinical practice, it has consistently demonstrated its ability to virtually eradicate sudden arrhythmic death, reducing it to less than 1% annually.

In MADIT, at an average follow-up of 27 months, there were only 3 deaths for the ICD limb classified as "primary arrhythmic" versus 13 deaths in patients randomized to drugs. The ICD results in MADIT are virtually identical to its performance in patients with a history of sustained, symptomatic VT/VF; similarly, the amiodarone results parallel those reported historically.

In this light, the MADIT results are not astonishing at all, but merely a strong confirmation of data we have known for many years. MADIT demonstrated powerfully that even patients with very poor LV function (average ejection fraction 0.26, and over 50% in New York Heart Association (NYHA) II/III heart failure) can have excellent overall survival through at least four years if protected against sudden death.

MADIT honed in on a homogeneous, high-risk target population, focused on a single important question, had a low cross-over rate, and had zero perioperative mortality. However, the design of the study was merely one of convenience. To be sure, one should avoid gross overgeneralization of any results, but the very idea that the strictly defined population which was selected was the only one in which the trial would have worked seems unconvincing, especially in light of the powerful outcome.

Thus, a second important challenge to MADIT concerns just how far its results can be applied to patients beyond those explicitly meeting the inclusion criteria for the study. It is already well-established that 80% or more of patients with sudden death and/or VT have coronary artery disease, most of them with previous myocardial infarctions. Compared to these, the MADIT patient has the same underlying disease and the myocardial scars known to be the site of origin of most reentrant VT.

In all important demographic characteristics (i.e., age, sex, previous revascularization), the MADIT patient is a

mirror image of most coronary patients with a history of VT/VF.³⁴ The degree of impairment of left ventricular function, the strongest predictor of VT/VF, is generally considerable in patients presenting with overt ventricular tachyarrhythmias, and such was the case for the MADIT cohort.

Since the efficacy of the ICD in such patients is not an unknown, the real question is whether antiarrhythmic drugs, in particular, amiodarone, might not perform just as well. This is in no way substantiated by available pertinent data. A striking finding in the recently completed "European Myocardial Infarction Amiodarone Trial" (EMIAT) was the abject failure of amiodarone to impact overall survival.³⁵

Why should these drugs suddenly do so much better in protecting the previously asymptomatic patients with a history of VT/VF enrolled in MADIT than they did in patients with previous cardiac arrest and asymptomatic VT/VF?

As for the ICD, there is significant data from multiple large series demonstrating its ability to provide secondary prevention from fatal VT/VF. At least in terms of protection against sudden death, there is no sound argument not to expect the ICD to outperform amiodarone or other antiarrhythmic drugs, whether for primary prevention, as in MADIT, or for secondary prevention.

Therefore, the only remaining outcome question concerns the concept of competing risks, whether the rate of heart failure death will, in effect, nullify the ICD's sudden death superiority. But, this important question depends only on the population being treated and the length of time, and is quite independent of whether the antiarrhythmic agent is amiodarone or the ICD. If the competing risks dominate, for a given patient or a given population, then neither agent can grossly improve survival. Given to patients whose risk is largely arrhythmic, the agent which better protects against sudden death must contribute more strongly to enhanced overall survival.

The only caveat relating to competing risk relates to patients excluded. Although the MADIT patients were significantly impaired functionally and anatomically, the study did exclude patients in NYHA class IV, those with evidence of active ischemia, and in general, those judged to have a life expectancy less than one year. Thus, it would not be realistic to assume that exactly the same results would occur with coronary artery disease patients whose clinical status is worse than those that were entered into the trial.

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Incidental detection of cystic neoplasms of the pancreas

Aaron D. Gorin and Jonathan M. Sackier, M.D., F.R.C.S., F.A.C.S.

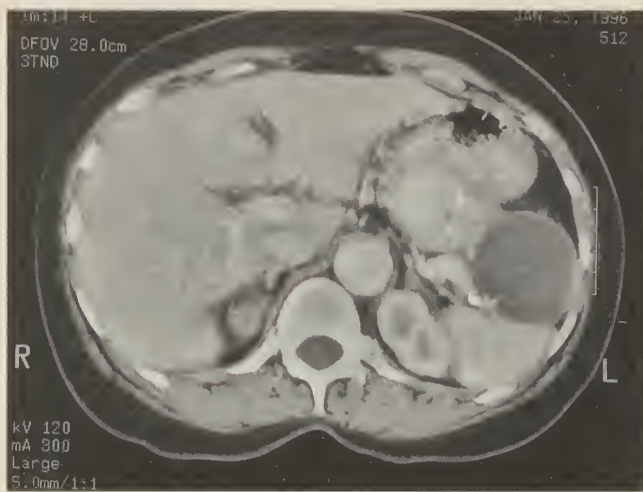
From the George Washington University School of Medicine, USA where Mr. Gorin is a fourth-year medical student and Dr. Sackier is associate professor of surgery and director, Washington Institute of Surgical Endoscopy.

ABSTRACT: Cystic neoplasms of the pancreas are rare, accounting for less than 1% of all pancreatic tumors.¹ Since the advent of computerized tomography (CT), an increasing number of these lesions are being discovered incidentally. Compagno and Oertel² were the first to thoroughly describe and differentiate the benign serous cystadenoma from the potentially or overtly malignant mucinous cystadenoma/cystadenocarcinoma spectrum. At present, our ability to definitively differentiate between these two classes of cystic neoplasms is limited. Because of this, controversy exists as to their appropriate surgical management. A case report is presented and followed by a review of the literature on incidentally detected cystic neoplasms of the pancreas.)

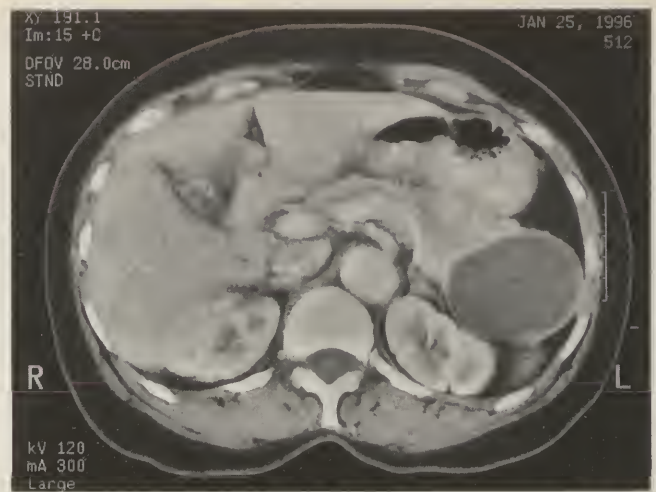
Case Report

A 57-year-old African American woman presented with a five-year history of recurrent microscopic hematuria often associated with urinary tract infections. An initial ultrasound and cystoscopy revealed no abnormalities. A repeat ultrasound five years later revealed a calculus in the calix of the left kidney which was thought to be the cause of her hematuria. In addition, a 2.8 cm round mass was found in the right adrenal gland. On CT, this lesion was thought to represent a benign non-functioning adenoma requiring no further workup.³ CT scan also revealed a 10.0 cm x 6.0 cm x 5.0 cm cystic mass in the left upper quadrant with calcification in its thickened wall (**Figure 1**). This was thought to represent a lesion in the tail of the pancreas.

At laparotomy, a large cystic structure, measuring approximately 10.0 cm in diameter, was found arising from the tail of the pancreas and



A



B

Figure 1. CT scan showing mucinous cystadenoma of the tail of the pancreas. Note its proximity to the spleen (A) and to the left kidney (B).

adherent to the splenic capsule (**Figure 2**). Distal pancreatectomy and splenectomy were performed without intraoperative histopathological differentiation between what was presumed to be either a mucinous or a serous cystic neoplasm of the pancreas. The patient had an uncomplicated recovery and was discharged eight days post-operatively.

Gross pathology revealed a 9.0 cm x 6.0 cm x 4.0 cm cystic mass with a white-tan capsule wall measuring up to 0.2 cm in thickness. The cyst was found to be unilocular and to contain thin golden fluid which was not analyzed. Focal areas of calcification within the wall were noted. Histological examination revealed a unilocular cyst measuring 9.0 cm in its greatest dimension. It was lined by a single layer of cuboidal to tall columnar cells, with occasional mucus cells present (**Figure 3**). A discontinuous layer of myoepithelial cells was present beneath the epithelial lining. The cyst was found to be partially denuded. Although no parenchymal cellular atypia was found, mild hyperplasia was noted in a nearby large pancreatic duct without atypical cellular changes. The remainder of the exocrine and endocrine pancreas showed no significant histological abnormalities. Based on these findings, the diagnosis of mucinous cystadenoma was made.



Figure 2. Intraoperative picture. Note the adherence of the tail of the pancreas to the splenic capsule.

Discussion

On macroscopic examination, mucinous cystic neoplasms of the pancreas usually appear as multilocular tumors with smooth surfaces. They often consist of a single macrocystic mass containing usually less than six cysts each with a diameter greater than 2 cm. Although their average diameter is 8 cm to 10 cm, large tumors of up to 25 cm are occasionally found. Pericystic inflammation is rare, and thus, adherence to extrapancreatic structures is not often seen at laparotomy. The most striking feature of these tumors is microscopic heterogeneity. While many areas contain a simple tall epithelium, cuboidal-appearing epithelium may also be seen in as many as one third of cases⁴, thus making it difficult to distinguish from the benign serous cystic neoplasm. In addition, myoepithelial cells may occasionally be found. Papillary invaginations, multiple areas of atypia, dysplasia, carcinoma in situ, and overtly invasive carcinoma may be discovered within the same lesion. Areas of denuded epithelium may also be found,⁵ which may make differentiation from a pseudocyst difficult. The epithelial cells usually express mucin and may stain for CEA, somatostatin, or serotonin, which suggests an origin from ductal cells. On analysis of the cystic fluid, mucin, increased viscosity, and increased concentration of tumor markers such as CEA or CA19-9 have been found to be of help in distinguishing these tumors from their benign serous counterparts.^{6,7} Although many mucinous cystic neoplasms may appear grossly and histologically benign, most authors agree with Compagno and Oertel that given sufficient time, virtually all of these tumors will form foci histologically identifiable as carcinoma. Thus, all mucinous cystic neoplasms are potentially malignant and must be resected.

Serous cystic neoplasms of the pancreas were described by Compagno and Oertel⁸ as being composed of innumerable tiny cysts with a honeycomb appearance on cross



A



B

Figure 3. Histological sections of mucinous cystadenoma of the tail of the pancreas. Note the areas of cuboidal-appearing epithelium (A) and simple tall mucin-containing columnar epithelium (B).

section. They are typically large (mean, 11 cm; range, 1.0 to 25 cm) and encapsulated. Serous cystic neoplasms are usually found in the head of the pancreas, while their mucinous counterparts tend to be seen in the body and tail.⁹ These tumors have a thin wall which may be translucent. Similar to the mucinous form, these tumors usually lack pericystic inflammation, and adherence to surrounding structures is rare. Histologically, the individual cysts are lined by a glycogen-rich, low-cuboidal epithelium without mucin production or evidence of cellular atypia or dysplasia (**Figure 4**). Areas of discontinuous epithelium may occasionally be found.⁵ A fibrovascular stroma usually separates the cysts, and a characteristic "sunburst" radial stellate scar pattern, may occasionally be seen on CT if this stroma becomes calcified (**Figure 5**). Serous cystic neoplasms seem to have their origin in the centroacinar cell¹⁰, which may explain their peripherally based location within the pancreatic parenchyma. These tumors do not stain for mucin or CEA, but do stain positively for periodic acid-Schiff, diastase-sensitive intracytoplasmic glycogen, which prompted their former description as "glycogen-rich."⁷ The cystic fluid is usually clear and without mucin.

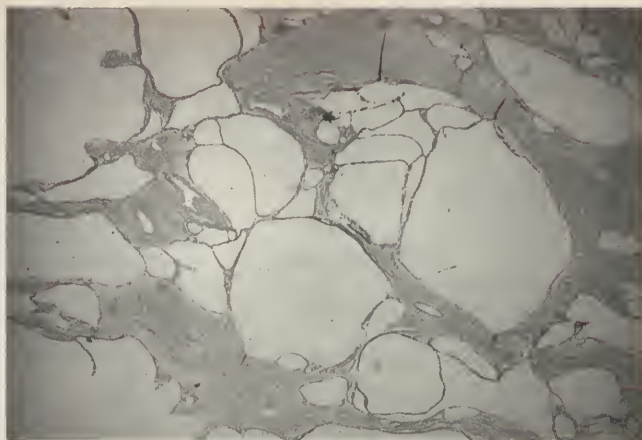


Figure 4. Representative histological section of serous cystadenoma of the head of the pancreas. Note the multiple cystic areas lined with a low-cuboidal epithelium.

(Courtesy of Kenneth Sisco, M.D.)

Serous cystic neoplasms have been described as having little or no malignant potential.⁸ Although malignant transformation has recently been documented,¹¹ it represents an extremely rare case and the original description by Compagno and Oertel still holds true.

The case presented above highlights an important decision that must be addressed: What is the appropriate surgical management of a cystic neoplasm of the pancreas found incidentally? Most authors now feel that all cystic neoplasms of the pancreas should be resected. This is based on the notion that, as of yet, we are not able to definitively differentiate serous cystic neoplasms, presumed to be benign, from their malignant, or pre-malignant, mucinous counterparts. Unilocular serous cystic tumors,¹² as well as multilobular, seemingly microcystic mucinous tumors, have both been described. Preoperative needle biopsy and intraoperative frozen section pathology are not absolutely diagnostic. This is due to the high frequency of cuboidal-appearing epithelium seen with mucinous cystic neoplasms,

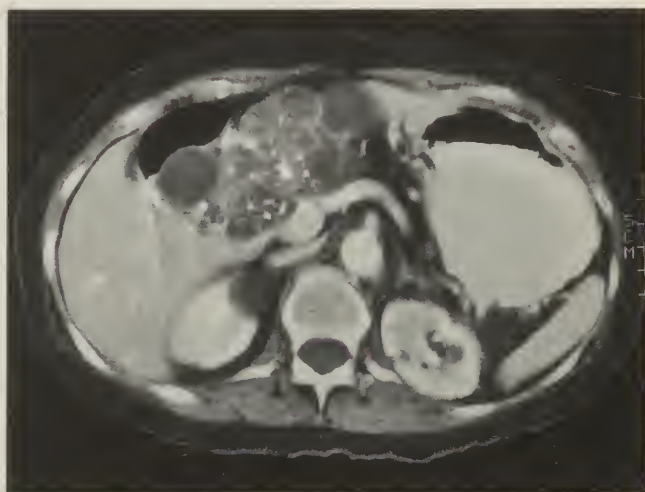


Figure 5. Representative CT scan of serous cystadenoma of the head of the pancreas. Note the central stellate calcification.

(Courtesy of Harendra Rupani, M.D.)

as seen in our case, as well as the possibility of a serous cystic tumor with a discontinuous epithelium. In addition, needle biopsies carry a small risk of complications, which includes pancreatitis and tumor seeding along the needle track if the cyst is malignant.¹³ The location of the cystic tumor may also offer only minor diagnostic assistance. As stated earlier, while mucinous cystic neoplasms usually occur in the body and tail of the pancreas, and the serous tumor in the head, the reverse is often seen. Due to the small short- and long-term morbidity and mortality associated with a distal pancreatectomy, as well as the reasons stated above, it would seem, as pointed out by prior authors,⁹ that resection of a cystic neoplasm in the body or tail of the pancreas is warranted without attempts at histological differentiation.

Cystic lesions of the head or uncinate process of the pancreas will require a Whipple-type pancreatoduodenectomy. As discussed earlier, preoperative or intraoperative histological diagnosis is of no diagnostic value. In patients whose tolerance of such an extensive procedure is in question, the risks of nonresective treatment, which include chronic pancreatitis¹⁴ and slow progressive enlargement with the eventual development of obstructive symptoms,⁹ must be weighed against the risks of the surgery itself. A palliative biliary or duodenal bypass may be indicated in patients with a high operative risk who have already begun to experience obstructive symptoms. However, with the recent decline in mortality after the Whipple procedure,¹⁵ most proximal pancreatic cystic neoplasms found incidentally are best treated by resection.

Because of the occasional areas of denuded epithelium found in mucinous cystic neoplasms, differentiation from a pancreatic pseudocyst, which is usually not an indication for subtotal pancreatectomy, may be difficult. In general, cystic neoplasms will not be associated with pericystic inflammation or adherence to adjacent structures. In addition, these tumors usually consist of multiple cysts. This is in contrast to pseudocysts, which are usually unilocular and show pericystic adherence. However, in the case of our patient, the cyst was unilocular and revealed pericystic adherence to the spleen. In addition, the preoperative CT scan revealed a unilocular cyst with a thick wall, which is also consistent with a pseudocyst. Although the final diagnosis in this patient was a mucinous cystic neoplasm of the pancreatic tail, a pseudocyst was certainly considered in the differential diagnosis.

Conclusion

Although unilocular pancreatic cystic neoplasms are quite rare, their incidental finding can pose somewhat of a quandary. Because of the diagnostic uncertainty in differentiating serous from mucinous neoplasms of the pancreas, and because of the very favorable outcomes of pancreatic surgery, subtotal pancreatic resection (along with splenectomy, if technically necessary) is recommended without intraoperative histological differentiation for all cysts located in the

body or tail of the pancreas discovered in this manner. In our case, the presenting findings of pericystic inflammation and adherence, a thick-walled cyst seen on CT, and its unilocularity, a pancreatic pseudocyst, which rarely requires pancreatectomy, was strongly considered. However, because of the patient's lack of predisposing factors (i.e., pancreatitis, alcoholism, trauma), combined with the significantly low mortality and morbidity associated with a distal pancreatectomy, the cyst was resected. In cases where a cystic lesion of the pancreatic head is found incidentally, an alternative management course may be indicated^{9,13}. However, the large majority of these cases are best treated by resection.

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Spontaneous mediastinal hemorrhage: a case report with a review of the literature

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ABSTRACT: *A patient on chronic hemodialysis presenting with shortness of breath and dysphagia was found to have massive hemomediastinum. A review of the world's literature prompted by this case reveals that this rare entity can be classified into three general groups: (1) hemomediastinum secondary to underlying bleeding disorder, (2) hemomediastinum secondary to hemorrhage into a mediastinal organ or gland, without underlying bleeding disorder, and (3) idiopathic hemomediastinum, without underlying bleeding disorder. Therapy depends upon the underlying etiology and the severity of symptoms.]*

Introduction

Non-traumatic mediastinal hemorrhage is an unusual clinical event. The etiology of this entity may be spontaneous, but usually is the sequela of an underlying clotting dysfunction or hemorrhage into mediastinal organs. We recently encountered a patient on chronic hemodialysis in whom a bronchogenic cyst was found in the midst of the mediastinal hemorrhage. The diagnosis may be based on history and appropriate imaging. Surgery may be required for diagnosis and/or relief of mediastinal compression.

Case Report

A 42-year-old male chronic dialysis patient was admitted to Prince George's Hospital Center with a two-day history of increasing substernal chest pain, dysphagia, and shortness of breath. The pain radiated to the jaw and did not improve with the administration of sublingual nitroglycerin. His medical history was notable for hypertension and end-stage renal disease secondary to Alport's syndrome. Past history included a



Figure 1. PA chest demonstrates mediastinal widening.

kidney transplant in 1976 that was rejected within two months. He had been on chronic hemodialysis since that time. Pertinent physical findings included hypertension and minimal pulmonary basilar rales. A chest film revealed mediastinal widening (**Figure 1**). A computed tomography (CT) scan (**Figure 2**) of the chest confirmed the mediastinal widening with tracheal and major bronchial compression. These findings, consistent with mediastinal hemorrhage, suggested the possibility of aortic dissection that prompted an aortogram (**Figure 3**). Over the next two days the patient became increasingly short of breath. With a diagnosis of mediastinal hemorrhage of unknown etiology, the patient was explored under local anesthesia via a left parasternal mediastinotomy. A large hematoma was found contained within the mediastinum. Blunt dissection of this hematoma resulted in the sudden appearance of an odorless thick

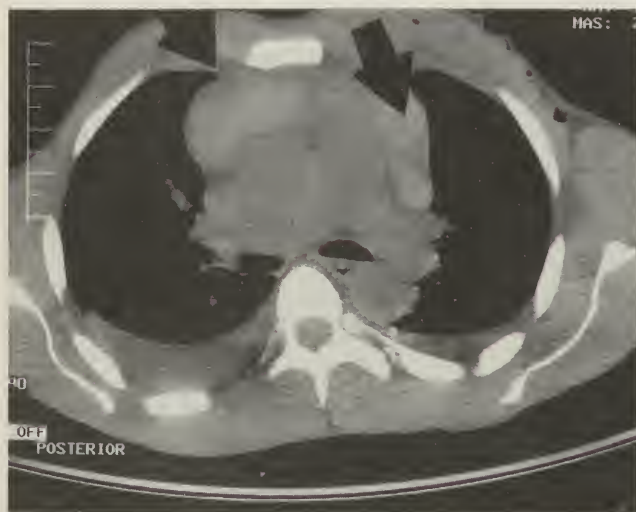


Figure 2. CT of chest. There is posterior displacement of the trachea by a large anterior mediastinal density. The base of the heart is posteriorly displaced. The air-filled esophagus is seen behind the left main bronchus. A right pleural effusion is present.



Figure 3. Normal aortogram.

yellow fluid. Cultures were taken, and abnormal tissue in the region of the para-ascending aortic area was excised. Permanent section of this tissue revealed columnar epithelium consistent with a bronchogenic cyst. The entire anterior mediastinum was thoroughly irrigated, and then the pleura to the left hemithorax was opened for post-operative drainage via a chest tube. The post-operative course was uneventful with the patient discharged seven days later. A follow-up chest film taken four months later shows complete resolution of the hemomediastinum (**Figure 4**).

Review of the Literature

Including the present patient, we found 39 cases of this disorder in the world's literature. Four reports were of two patients; the remainder were single case reports. The age range was 3 to 76 years (mean of 47 years), with the age unstated in 2 cases. Twenty-four patients were male and 12 female, with the sex unstated for 3 patients. We grouped the patients into three general types: patients with underlying bleeding disorders (n=19), patients who bled into mediastinal organs, glands, or cysts without bleeding disorders (n=9), and idiopathic patients without bleeding disorders and hemorrhage into or around a mediastinal gland or cyst. Five patients in the group died. Twenty-one patients required 22 operative procedures. These included 3 sternotomies, 7 thoracotomies, 7 mediastinotomies or mediastinoscopies, 3 tracheostomies, 1 thyroidec-

tomy, and 1 needle aspiration. The superior mediastinum was primarily affected in 19 patients, the anterior in 7 patients, the anterior-superior in 1 patient, the superior-posterior region in 1 patient, the posterior mediastinum in 3 patients, and unspecified mediastinum in 8.

Discussion

Spontaneous mediastinal hemorrhage is a rare and portentous clinical event. When encountered, the symptoms may warrant immediate surgery or, in some instances, supportive therapy may be the appropriate management. The etiology of the condition frequently remains obscure unless one is aware of the entity.

The most common etiology of mediastinal hemorrhage is non-penetrating trauma, specifically, rupture of the descending aorta. Other conditions related to mediastinal hemorrhage and the aorta include dissection and sequelae related to surgery or angiography.

Non-aortic mediastinal hemorrhage can be classified into three general groups: 1) hemomediastinum secondary to bleeding disorders, 2) hemomediastinum secondary to hemorrhage into mediastinal glands or cysts without bleeding disorder, and 3) idiopathic hemomediastinum without bleeding disorder (Tables 1,2,3). Our patient is an example in which two of the more common preexisting conditions were present: chronic hemodialysis, with its attendant clotting dysfunction, and a mediastinal bronchogenic cyst. The bronchogenic cyst in this case, however, was an incidental finding. The exact

TABLE 1. Spontaneous mediastinal hemorrhage with abnormal clotting or fibrinolysis

Author	Year	Age/Sex	Surgery	Area of Bleed	Outcome
<i>Patients on Dialysis</i>					
▼ Ellison	1981	71/M	Mediastinotomy	Anterior Mediastinum	Survived
▼ Ellison	1981	32/M	Thoracotomy	Mediastinum	Survived
▼ Present Case	1996	42/M	Mediastinotomy	Anterior Mediastinum Bronchogenic Cyst	Survived
<i>Patients on Anticoagulants or Fibrinolytics</i>					
▼ Packer	1972	39/F	None	Superior Mediastinum	Survived
▼ Henschler	1975	31/M	Mediastinotomy	Superior Mediastinum	Survived
▼ Kaplinsky	1978	60/F	None	Mediastinum	Survived
▼ Turetz	1979	74/M	None	Anterior Mediastinum	Survived
▼ Abaskaron	1983	54/F	None	Superior Anterior Mediastinum	Survived
▼ Mazziotti	1983	Unknown	None	Mediastinum	Not stated
▼ Singh	1983	72/M	None	Mediastinum Thymic Cyst	Died
▼ Lawler	1984	66/M	Thoracotomy	Superior and Posterior Mediastinum	Survived
▼ Suddes	1988	48/M	None	Superior Mediastinum	Not stated
▼ di Pasquale	1990	53/M	Needle aspiration	Anterior Mediastinum	Survived
<i>Patients with Hemophilia</i>					
▼ Edmonds	1951	7/M	Tracheostomy	Superior Mediastinum	Died
▼ Pochedly	1968	3/M	Tracheostomy	Superior Mediastinum	Survived
▼ Jivani	1970	11/M	None	Mediastinum	Survived
▼ Bart	1972	17/M	None	Mediastinum	Survived
▼ Siefkin	1984	23/M	None	Posterior Mediastinum	Survived
<i>Patient with Familial Hemorrhagic Diathiasis</i>					
▼ Masuelli	1953	61/F	None	Superior Mediastinum	Survived

TABLE 2. Patients with bleeding into mediastinal organs or into abnormal structures

Author	Year	Age/Sex	Surgery	Bleeding into organ/gland Pathology	Outcome
▼ Capps	1934	50/M	None	Parathyroid adenoma	Died
▼ Sandor	1964	54/M	None	Thyroid goitre	Survived
▼ Berry	1974	63/M	Median sternotomy	Parathyroid adenoma	Survived
▼ Gatzinsky	1978	34/M	Thoracotomy	Intramural esophageal cyst	Survived
▼ Moskowitz	1980	12/M	Parasternal Mediastinotomy	Thymic cyst aplastic anemia	Died
▼ Moskowitz	1980	6/F	Mediastinotomy	Thymic cyst aplastic anemia	Died
▼ Prenger	1984	62/F	Sternotomy	Thyroid bleeding following I 131	Survived
▼ Fukuse	1991	Unknown	Thoracotomy	Thymoma	Survived
▼ Snyder	1990	62/F	Mediastinoscopy	Ectopic Thyroid	Survived

TABLE 3. Spontaneous mediastinal hemorrhage without underlying clotting dysfunction

Author	Year	Age/Sex	Surgery	Area of Bleed	Outcome
▼ Benedetti	1958	33/M	Thyroidectomy	Superior Mediastinum	Survived
▼ Epstein	1960	66/F	None	Superior Mediastinum	Survived
▼ Epstein	1960	76/M	None	Superior Mediastinum	Survived
▼ Marion	1968	47/F	Tracheostomy Sternotomy	Superior Mediastinum	Survived
▼ Hennessy	1970	54/Unknown	None	Superior Mediastinum	Survived
▼ MacDonald	1975	58/F	Thoracotomy	Posterior Mediastinum	Survived
▼ Hidalgo	1975	72/F	Thoracotomy	Mediastinum and L&R Pleura	Survived
▼ Culliford	1977	44/M	Thoracotomy	Mediastinum and L Pleura	Survived
▼ Stilwell	1981	75/M	Mediastinotomy	Superior Mediastinum	Survived
▼ Marchan-Carranza	1994	60/M	None	Superior Mediastinum	Survived
▼ Marchan- Carranza	1994	55/F	None	Superior Mediastinum	Survived

mechanism of the bleeding in spontaneous mediastinal hemorrhage, as well as the reasons why hemorrhage is limited to the mediastinum and which vessels are involved, is uncertain.

Diagnosis of mediastinal hemorrhage is radiographic; a widened mediastinum in an appropriate clinical setting. It requires an awareness of the existence of the entity. Symptoms (Table 4) include sudden retrosternal chest pain, dyspnea, dysphagia, cough, hoarseness, and dys-



Figure 4. Follow-up PA chest showing resolution of hemomediastinum.

phonia. These symptoms may be preceded by an episode of vomiting and retching. Physical findings of neck swelling, ecchymoses, and/or shock depend upon the location of the hemorrhage and rate of bleeding, often associated with a drop in hematocrit. Aortography may be urgently required to rule out aortic disease. Once the vascular catastrophe has been excluded, the differential diagnosis may be made in a systemic fashion, unless the compressive symptoms are life threatening.

As in every disease, diagnosis precedes therapy. If one is convinced of the diagnosis, surgery would not be warranted unless a significant physiologic disturbance is present. If the diagnosis is in doubt, exploration of the mediastinum should be performed. In the instance of mediastinal compression, drainage of the hematoma will be therapeutic as well as diagnostic.

TABLE 4. Signs and symptoms of spontaneous mediastinal hemorrhage

Symptoms

▼ Pain	27
▼ Dyspnea	19
▼ Dysphagia	13
▼ Hoarseness/dysphonia	8
▼ Choking	1
▼ Cough	10
(two with hemoptysis)	
▼ Vomiting	6
(one with hematemesis)	
▼ Stridor	4

Signs

▼ Neck Swelling	18
▼ Ecchymoses	14
▼ Shock	4

* These signs and symptoms were tabulated from 39 patients from 35 reports, including ours. Not every paper of "Spontaneous Mediastinal Hemorrhage" reported the signs and/or symptoms.

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46,XY,i(21q) identified by maternal serum screening

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ABSTRACT: Maternal serum screening for the detection of fetal Down syndrome has become widespread. Prenatal detection of fetal Down syndrome has important implications not only for management of the current pregnancy, but also for recurrence risk counseling for future pregnancies. We report a case of fetal Down syndrome due to an isochromosome 21q detected after maternal serum screening using alpha-fetoprotein and human chorionic gonadotropin indicated an increased risk for fetal Down syndrome in a 19-year-old pregnant woman. This confirms that maternal serum screening can detect fetal Down syndrome due to rare chromosome rearrangements and illustrates the importance of cytogenetic studies for provision of appropriate genetic counseling.

Introduction

The association of low maternal serum alpha-fetoprotein (MS-AFP) values and chromosomal aneuploidies was first demonstrated in 1984 by Merkatz and others.¹ Subsequently, the combination of maternal age and MS-AFP values provided prenatal screening for detection of fetal Down syndrome in pregnant women not previously known to be at increased risk.² More recently, it has been observed that maternal serum human chorionic gonadotropin (MS-hCG) values are increased in Down syndrome pregnancies. Wald and others² obtained a 56% detection rate of Down syndrome using maternal age, MS-AFP, and MS-hCG levels in women under 35 years of age. Additional studies have shown similar detection rates.³⁻⁵

A positive maternal serum screen does not diagnose chromosome abnormalities, nor can it differentiate between the various chromosomal abnormalities that result in the Down syndrome phenotype. Because

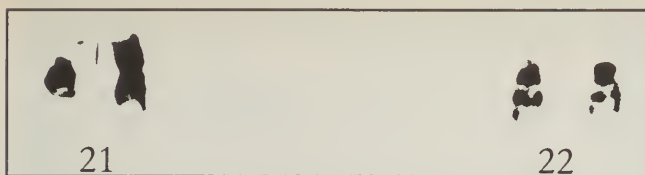


Figure 1. Partial fetal karyotype showing i(21q;21q).

individuals with Down syndrome due to a chromosome rearrangement are phenotypically indistinguishable from those with Down syndrome due to free trisomy, it is reasonable to postulate that cases due to chromosome rearrangements would be detectable by prenatal screening with maternal serum markers. One maternal screening program reported detection of four D;G or G;G translocations, but specific karyotypes were not given.⁶ We report a case of isochromosome 21 (i(21q)), the rarest rearrangement resulting in the Down syndrome phenotype, detected by maternal serum screening.

Case Report

The patient was a 19-year-old Caucasian pregnant woman whose medical and family histories were unremarkable. Her pregnancy had been uncomplicated. This was the first pregnancy for the 19-year-old father whose medical and family histories were also unremarkable. At 16 weeks gestation by dating and sonographic criteria, her MS-AFP level was 0.72 multiples of the median (MoM) and her β -hCG level was 3.79 MoM, indicating a risk for fetal Down syndrome of 1 in 83. By age alone, her fetal Down syndrome risk was 1 in 1180. After genetic counseling, amniocentesis was performed at 19 weeks gestation. Ultrasonography prior to amniocentesis revealed an abnormal fetal heart with no other fetal anomalies; endocardial cushion defect was confirmed by fetal echocardiography. The parents elected to terminate the pregnancy. Karyotype from amniocytes, fetal lymphocytes, and fetal fibroblasts was 46,XY,i(21q) (Figure 1); amniotic fluid AFP was 0.9 MoM. Cytogenetic analysis of parental lymphocytes revealed normal karyotypes.

Discussion

This case suggests that prenatal screening with maternal serum AFP and hCG is not limited to detection of Down syndrome due to non-disjunction, but can also ascertain cases of i(21q). The phenotypic characteristics of Down syndrome result from the triplication of a region of chromosome 21 including 21q22.3.⁷ In 3% to 5% of Down syndrome cases, triplication of 21q22.3 is the result of rearrangements involving chromosome 21. The rarest of these was previously thought to be a 21q;21q translocation. It has now been shown that the majority of such cases are due to isochromosome formation rather than translocation.⁸ Moreover, the origin of the i(21q) is equally derived from paternal and maternal meiotic errors⁸⁻⁹ and is not associated with advanced maternal age. Detection of these cases is particularly

important, as approximately 5% are inherited.¹⁰ For familial cases, the recurrence risk for Down syndrome is essentially 100%. The only possible conceptions are monosomy 21, which is non-viable, or trisomy 21. A history of multiple miscarriages or multiple Down syndrome offspring may be elicited in inherited cases. Use of donor sperm or ova may allow these couples to have chromosomally normal offspring.

For this couple, the recurrence risk is essentially zero because the isochromosome is not familial. Had cytogenetic studies shown the more common 47,+21 karyotype, recurrence risk for aneuploidy in future pregnancies would have been approximately 1%.¹¹ This case confirms that maternal serum screening can detect fetal Down syndrome due to chromosome rearrangements and illustrates the importance of cytogenetic studies in providing appropriate genetic counseling.

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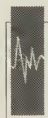
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Managed care and the physician-patient relationship: implications for peer review

The nature of the physician-patient relationship is changing. With ever more frequency, the physician's ability to make decisions about the care of the patient is inhibited by the intervention of other parties who have direct financial interest in the cost of that care. The introduction of third parties into medical decision-making redefines the care-giving process. As our understanding of the roles of physician and patient evolves, we must change our notion of the absolute character of the physician's responsibility to the patient, as well as our notion of the absolute character of the patient's vulnerability.

The traditional model

In the traditional model of the physician-patient interaction, the patient is regarded as absolutely vulnerable, placing all trust in a physician. By the Oath of Hippocrates, the physician swears his or her intent to act in the patient's best interest and forswears any behavior which would injure the patient or place the patient at unnecessary risk. The physician is regarded as dominant in this relationship, able to exert free will to make the proper moral decision and perform those acts which are in the best interest of the patient, while avoiding those acts which are harmful to the patient or which might be regarded as mainly selfish.

In the traditional view of the physician's role in guiding patient care cost is not considered, only the best care possible for the patient, often despite the cost. Inherent in this model is the expectation that the physician is an advocate for, and the patient is entitled to, the best care possible, not adequate care that may be less costly. The central point of this model is the patient's welfare, and, borrowing the notion of egalitarianism from our concept of equal justice under law, and from our strongly-held belief in fair play in a free market economy, it has been the physician's duty to see to it that the patient receives all the care from which the patient could benefit.

The managed care model

In the managed care model, the central focus is the cost incurred in caring for a population of patients. The

welfare of the individual patient, while important, is less of a driving force in this system than the welfare of the entire population of patients. Issues of care are always tied directly to issues of cost, and the goal for the physician is to adopt a treatment strategy for the patient which will be both adequate and cost-effective. Here the physician serves two masters: the patient and the managed care organization (i.e., the population of patients).

The needs of the patient are the same in this model as in the previous one—namely, to receive proper care. However, in the managed care model, the patient has made an arrangement with the managed care organization prior to the doctor-patient relationship. This arrangement, which is a contractual agreement to abide by the rules of the managed care organization, usually involves the intercession of a primary care physician between the patient and all other health care providers. The patient has agreed in his or her insurance contract that the primary care physician will decide whether, and under what circumstances, the patient may seek additional or secondary care. (In addition, the primary care physician may have a financial incentive to limit procedures, referrals, and other costly care, since he places his own future with the managed care organization in jeopardy if his patients' care is too costly. In some cases, he can benefit by direct financial rewards from fee withholds if, at the year's end accounting by the managed care organization, he demonstrates an economical pattern of practice.) Implicit in this agreement is the goal of reducing the cost of the patient's medical care, although the patient may not be explicitly advised of this goal by the managed care organization. Instead, the patient is usually advised that the goal is to enhance the quality of his or her care by reducing unnecessary tests and procedures.

The physician has also made a prior agreement with the managed care organization. In order to receive referrals from that organization, the physician has signed a contract to provide care to a group of patients, usually at a predetermined (and reduced) rate, and with the additional constraint of certain limitations on the ability to order tests, utilize laboratory services, perform procedures, and do surgery without

the approval of the managed care organization or its agent.

Of the physician or the patient, few would argue that the physician is more knowledgeable about the constraints faced in the managed care model. In fact, it is widely observed by physicians that patients are often surprised at the limitations imposed on physician behavior by managed care organizations, especially at times when patients neglect to obtain referral forms and cannot be accommodated in specialists' offices, or when patients learn that familiar doctors are not members of certain plans. Physicians, on the other hand, cannot be surprised at any of these features of managed care, since those who participate have had the opportunity to read and assent to written contracts which describe these provisions in detail.

The doctor-patient relationship under managed care

The doctor-patient relationship in the managed care model is complex. Both the doctor and the patient share the overall goal of patient care. However, the kind of care which the patient receives is not similar in all respects to that given in the traditional model. In the managed care model, both the doctor and the patient have made contractual agreements with a third party, the managed care organization, to behave in certain ways and to abide by certain rules, mainly to economize on the cost of care. From the doctor's perspective, the care should be adequate to the needs of the patient. From the patient's perspective, the care should not be so excessive as to inflate the cost of that care, since it was the patient's intent to pay less for the managed care insurance contract, while preserving an acceptable level of quality.

In the managed care model, both the physician and the patient must have different expectations from each other than they do in the traditional model. The physician plays a less dominant role; he or she is less able to act on his or her own, and becomes more of an advisor and a partner with the managed care organization. Said another way, the managed care organization emerges as a full partner in the care of the patient, with the responsibility to act prudently in the interest of the patient. Unable to act on his or her own to order tests and perform procedures without the approval of the managed care organization, the physician shares with the patient a dependence on the organization's cooperation in the execution of the care which the physician has planned. While it is possible to theorize

that the physician is free to carry out any therapeutic plan to which the patient assents, few patients are financially able to act independent of their insurance company's reimbursement decisions, so that a lack of authorization by the managed care organization to reimburse care suggested by the physician becomes tantamount to a denial of that care. In the managed care model, with the physician and the organization acting as partners in the delivery of the care, the patient is unable to receive care unless both partners cooperate to provide it.

Unlike the traditional physician, the managed care physician can be said to have less latitude in making medical care decisions. He may be able to advise the patient with freedom, but he or she is not able to treat the patient with total freedom, since most, if not all, of his or her medical decisions require the use of facilities, drugs, devices, and other accouterments of medical practice which create costs, and which, for the most part, fall within the category of "covered medical services" defined in the contract with the managed care organization. The physician has agreed to rely on the managed care organization for the reimbursement of "covered services," and he further agrees not to bill the patient under any circumstance. Thus, even if the patient wished to make a private contract with the physician or health care facility, it would not be feasible because that care would almost certainly fall within the definition of "covered medical services," precluding the physician or any other entity under contract with the managed care organization from billing the patient directly. A denial by the managed care organization to authorize reimbursement for care becomes identical to the denial of the physician/managed care organization partnership to provide care.

In the managed care model the physician cannot be held solely and individually responsible for providing all care to the patient, but instead, the physician and the managed care organization must be regarded as jointly responsible. To conclude otherwise would be to deny the important prerogative exercised by the patient in the managed care contract, which both limits premium cost and the independent actions of the physician. If the patient agrees to these contract provisions, the traditional physician-patient relationship does not exist. This is not to say that the patient should be satisfied with lower quality care, only that the patient must recognize that the physician is no longer able to act as a free and independent provider of that care. The physician may be the patient's advocate, but within the limits imposed by the managed care contract.

Implications for peer review

The redefined relationship between the managed care physician and patient creates a challenge to the process of peer review. In the traditional model, the assignment of responsibility for the patient's care is usually straightforward, since that care is almost always under the direct supervision and authority of a licensed physician. However, in the managed care model, the physician acts with less independence and with less absolute authority to direct the patient's total care, limited by the applicable rules and contractual limitations of the managed care contract agreed to by both the physician and the patient. No longer is it possible to assume *prima facie* that all deviations from the standard of care are solely due to the physician's omissions or commissions. Each instance of substandard care must be analyzed anew, so that those responsible can be properly identified. In some cases, the physician's acts themselves will be identified as the cause of substandard care. In other instances, the way in which the managed care organization functions to deliver care to its patients will be responsible.

Cases in which substandard care results from deficiencies in the managed care organization's operations create ambiguities in assigning responsibility. The Board of Physician Quality Assurance (BPQA) has jurisdiction over licensed physicians and certain other types of individual health providers, not over organizations. From the vantage point of providing disciplinary action for substandard medical care, it becomes necessary to identify individual licensed health care providers as the focus of peer review investigations. Indeed, the very nature of "medical care" must be widened to include the actions of licensed health care providers with the scope of health care organizations. Not only must peer review investigations look into the physician's role in providing care to the individual patient, but also into the role played by those physicians who are responsible for the creation of the rules and procedures of the managed care organizations, rules and procedures to which both their insured patients and their provider physicians are bound by contract.

Peer review investigation remains focused on individuals, not organizations, in concord with the jurisdiction of the BPQA. Case analysis will always be stimulated by the need to decide whether a breach of the standard of care occurred. However, in cases

involving physicians working within the scope of a managed care organization, this question must always be asked: "Was the quality of the care affected adversely by the limitations placed on that care by the managed care organization's rules or procedures, and was this the principal reason for the breach of the standard of care?" If so, then it becomes necessary to try to identify the licensed health care providers responsible for the formulation of the rules and procedures of the managed care organization. These individuals can then become the focus of further investigation which concentrates on the apparent causes for the substandard care, including the effect of the managed care organization's rules and procedures on the actions of those physicians providing the care.

The peer review report and the actions of the BPQA

Whenever changes occur in the delivery of medical care, changes must be made in the way we assess the quality of that care. The traditional concept of the physician-patient relationship is no longer fully applicable in the managed care model, and with the sharing of the provision of care between the physician and the managed care organization, we are called upon to redefine who is the actual responsible provider of care. The peer review process must be able to focus on individuals, because of the jurisdiction of the BPQA. At times, the responsible provider will be identified as the physician caring for the patient. At other times, the managed care organization's rules and procedures themselves may be identified as the reason for a breach in the standard of care. In such situations, it will become necessary to look more closely at those individuals who create these rules and procedures, particularly when they are created by licensed physicians subject to the BPQA's authority. It will then be up to the BPQA to struggle with these same issues from its own vantage point and to make its own policy as to how best to formulate the appropriate disciplinary action.

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Book review editor:
Chris Papadopoulos, M.D.

The CD-ROM contains the complete text and illustrations of the sixth edition of Kaplan's and Sadock's *Comprehensive Textbook of Psychiatry/VI* (CTP/VI), plus all the terminology entries in Frank Ayd's *Lexicon of Psychiatry, Neurology, and the Neurosciences*. The new nosology of the fourth edition of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) is a component of the CTP on CD-ROM database by virtue of its integration throughout the content of CTP/VI.

CTP on CD-ROM gives the user rapid and efficient access to information anywhere within the text, tables, and references contained in the over 2800 pages in CTP/VI. Similarly, one can consult the Lexicon for authoritative definitions of terminology used in psychiatry, neurology, and the neurosciences. They can be searched individually, as separate resources, or simultaneously as one encyclopedic reference.

The search and retrieval interface used for CTP on CD-ROM is Hierarchical Data Technology (HDT), which was designed and developed by Teton Data Systems of Jackson, Wyoming. HDT is a powerful full-text tool for electronic publishing on optical or magnetic media. Speed and simplicity of use characterize the search and retrieval functions. It has three useful functions named "Smart Match," "Match Summary," and "Precision Control." These allow retrievals that can be broad but will identify invalid information, and others that can be narrow and possibly allow the user to miss important information. One can scroll through the text of information retrieved and use hyperlinks for tables, illustrations, and cross-references. In addition, the user can place

retrieved information in bookmarks, sticky notes, and print the text. All these procedures are relatively easy to use and the Help menu is excellent.

Compared to two other CD-ROMs, American Psychiatric Electronic Library (APEL) and SilverPlatter's subset of the Medline database for Psychiatry, CTP on CD-ROM did well on an examination of post-traumatic stress disorder (PTSD) and psychotropic medications, but provided too many hits on low precision and zero on high precision. It totally failed to find information relating to Risperidone to neuroleptic malignant syndrome, whereas the other two programs identified this relationship. Basically, CTP on CD-ROM failed to retrieve material relating to recent information. This is not surprising as textbooks rapidly become outdated. In contrast, APEL and Medline are constantly updated and, therefore, more likely to retrieve recent information from an appropriate search.

This CD-ROM is the same price as the textbook, although the Lexicon comes free with the CD-ROM. It can be loaded into a Windows 3.1 or Windows 95 operating system and there is an "About CTP on CD-ROM" window that provides details about the content and various features by which you can search and retrieve information. Its weakness as a CD-ROM is the result of its becoming outdated. This is obviously a flaw for a tool that has as its greatest strength search and retrieval power. It is also not a useful tool to be utilized as an alternative text to paper.

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PRACTICE ISSUES

Ten rules for the road

A couple of years ago, Jim Sheehan, Assistant United States Attorney for the Eastern District of Pennsylvania, penned a quick guide titled "*Ten Ways to Meet The OIG*" (Office of Inspector General). For years, I have relied upon these "ten ways" in my seminars and training classes. Usually they elicit some light chuckles, and then, as reality sets, in the concern becomes evident on the participants' faces.

I will take each of the rules and explain why they were singled out.

1. **Delegate all billing responsibilities to your office staff. Never ask what they are doing. Never have activities reviewed by outside professionals (i.e., accountants, billing experts). Let them know that all you care about is the bottom line. Never ask how they translate your services to a claim.**

When using in-house staff to file your claims, be sure to hire supervisors who are professionally trained in medical billing. This supervisory personnel should be able to provide you with a litany of *current* seminars and meetings related to medical claims administration that they have attended. Continue to send them for additional training to seminars put on by national organizations such as Medical Group Management Association (MGMA), American Academy of Procedural Coders (AAPC), Professional Association of Health Care Office Managers (PAHCOM), etc.

Create a corporate integrity program and follow it. (See *Md Med J*, 45;10:861–865.) Close the office three hours early, buy a deli tray, and sit down with staff in an open meeting every quarter, or semi-annually, and allow them to speak freely without retribution. This allows a free exchange of ideas; you will find out many things that happen on a daily basis that you are currently unaware of, but need to know.

I have yet to attend one of these meetings and not have physicians come out amazed at what they have learned. If you have more than one office, gather everyone to a single location for this activity. Sit back and let them run the show. You are but a moderator until the end. Let it freely flow. What you learn will be substantial. What you *may* learn is how different each office truly is and how each handles situations with a different philosophy that may not mirror your own.

This will give you an opportunity to discuss implementing a corporate integrity program and discuss your expectations of them in helping maintain compliance. Explain that they have a stake in your practice being the best it can be both clinically and administratively.

2. **Hire a billing service to handle all your billing; pay them on a percentage basis. Never ask what they are doing and never have activities reviewed by outside professionals (i.e., accountants, billing experts). Use a billing service with a track record of substantial recoveries in every office in which they have worked.**

It is *illegal* to pay a billing service a percentage based on any Medicare collections. It is felt that this would induce the billing service to upcode your claims to the carrier, thereby increasing revenue (yours and theirs). I posted this to the Part B Listserv on the internet and the discussion continued for weeks. I was warned of this phenomena last year by a Medicare fraud investigator. I was also told that emphasized attention was being paid to any billing service or consultant who used phrases like "Maximum Revenue" or "Increased Revenue."

You should have an accountant or consultant with a strong healthcare background conduct an annual independent review of billing procedures whether your staff files your claims or you use a service. This should be done for your own protection. No matter who bills your claims, whether a service or staff, *you* are responsible if they violate standards. You will not be successful in trying to defer blame to the service or staff.

3. **Order large numbers of ancillary tests (blood, Doppler, MRI), to be performed by entities in which you have a financial interest or who "rent" space or otherwise pay you, based in part on referrals.**

A few years ago Congressional Representative Fortney "Pete" Stark ordered a study that found that physicians with a financial interest in an ancillary service had a strong tendency toward ordering more tests directed to that facility. When a more extensive study was done by Stark in Florida, the results were even more dramatic. It was then that Stark I was passed, stating that you must advise your patients that you are sending them to a facility in which you have a financial interest.

Stark II went further and there was a potential Stark III that did not pass. Stark II required you to disclose to the patient your financial interest in any facility where you are referring that patient.

4. **Bill for services performed in hospitals by residents. After all, you probably talked to them about the case at some point, even if your name is not on the chart.**

Hospitals are reimbursed for services provided by residents as part of the Graduate Medical Education (GME) funds in the hospital rate structure (costs reports). If the resident provides the service then no one gets billed for that service; it is considered already paid. Clinical Practices of the University of Pennsylvania (\$30 million) and Thomas Jefferson (\$12 million) have just paid fines for this and other violations. Now the OIG has announced that all 125 teaching hospitals will be audited for this practice. Keep in mind, funds for the fines involve physician dollars at some point, as the physician ultimately benefited.

If, however, you supervised the resident and have *extensively* documented all of your conversation and concurrence, then you should bill. You cannot countersign, or sign, read,

and agreed to, or I concur. You must detail each system discussed, your professional opinion, and your concurrence with the diagnosis and treatment in detail.

5. **Bill for hospital or nursing home visits when you have made no entry on the patient's chart. Or better yet, bill your in-hospital patients on a routine system (e.g., 5 days out of every 7 without actual records of patient visits).**

Nursing home fraud is rampant. Last year the President and the Attorney General launched Operation Restore Trust targeting nursing homes, home health and durable medical equipment (DME) suppliers. Do not "gang bang" or "gang visit" nursing home patients. Medicare can now track your services on a daily basis to see if you are saying that you are seeing more patients than you possibly can. They can now track your services relative to the number of hours in a day.

Make sure that you document all patient visits in the nursing home or hospital chart for all visits. Never "pattern bill," that is, don't bill on a cycle of seeing patients a set number of days out of a set number of days. Don't bill for "wave" visits (waving to the hospitalized patient from the door and asking how they are).

6. **Sign orders, prescriptions, or CMNs for patients you haven't seen recently. (The request probably wouldn't have come into your office unless the patient really needs it.)**

DME fraud is one of the United States Attorney's major fraud areas. Unscrupulous DME vendors will call your office in an attempt to get a certification of medical necessity (CMN) on behalf of a patient, so they may submit a claim to Medicare. Usually, this begins with an unsolicited phone call to a Medicare beneficiary with an offer of a free chair lift or special bed. The beneficiary hears the word free, the caller suggests symptoms, and the patient then (sometimes) authorizes the call to your office for the CMN which is mandatory to bill Medicare. Your records need to back up your action. When the government catches the DME provider, they trace the CMN back to you and want to see your documentation for authorizing the product.

Liability is yet another concern. Prescribing medications without a current medical history (including other medications that another physician may have prescribed) is risky both for the patient and your practice. If you haven't seen the patient recently, have the patient schedule a visit prior to any prescribing of medications.

7. **Bill for technical services performed by "employees" who "work" for you whenever you call their company. Or let a non-physician use your number to bill for services performed "under your supervision."**

"Under your supervision" refers to Medicare's "Incident to" rule. Under "Incident to" a physician may bill for services performed by his or her employee as long as they are incident to the physician's services and under the direct supervision of the physician (the physician must be in the office suite when

the service is provided). The physician must also provide the initial service to the patient. Recently, HCFA announced that it will allow a physician to bill for services for "leased employees" under the incident to rules. However, the incident to rules are in no way intended to allow a physician to bill for nurse practitioners or physician assistants in the hospital setting. Physician assistants in an emergency room can be billed when employed by the physician group.

8. **Ignore complaints from patients about bills. After all, it's not their money. Just have your secretary tell them insurance will pay for it and you'll write off the balance.**

Complaints from patients now must be turned over to the Medicare Fraud and Abuse Investigation Coordinator (MFAIC). Commercial carriers rely on patient complaints to help identify providers with potential fraud and billing problems. Most commercial carriers are members of the Health Care Anti-fraud Association and share information regularly over "troubled providers." Too many complaints about the same provider is an indication of bigger things and will certainly raise the scrutiny about your practice.

You already know that writing off copayments and/or deductibles is both a violation of all of your contracts and considered insurance fraud (see *Md Med J* 45;4:332-334).

9. **Write whatever Medicare or the insurer needs to hear to pay the bill. Nothing is ever routine or a check-up.**

This seems like an obvious missive, but you would be surprised. Never dictate medical records directed to the insurer's payment methodology. They may end up being words that become less than a gastronomic delight in a medical malpractice case. This includes how you code the diagnosis and any procedures performed. If and when audited, *never* change a word, a syllable, or a punctuation mark on a medical record. It is a crime and will only add to your problems. This is where the true benefit of *properly educated* staff ultimately shows through (see *Md Med J* 45;1:52).

If the service should be billed as routine or a check-up, call it that. The small amount of revenue created using "creative billing" is most certainly not worth it, and more important, morally wrong.

10. **Don't keep consult information or lab results in the same file as your patient records; you'll remember it was done if you really need it.**

Memorize this phrase (because I assure you the auditors know it by heart): "If it's not documented — It's not done." Plain and simple. You will never successfully argue otherwise. HCFA implemented new documentation guidelines for Evaluation and Management (E & M) codes over a year ago. If you don't have a copy of the guidelines, you can call your carrier or contact me and I will send them to you.

I will take this a step further; keep your clinical and patient financial records separate.

C. SHERWOOD HARRIS, CCAM

Mr. Harris is the executive director of Keep InforM.D., (410-494-0694), a health care news and legislative services company that produces three newsletters for the physician community. Keep InforM.D. can also be found on the internet at www.access.digex.net/~informed. ■

Question

Blue Shield of Maryland recently published their coverage guidelines for bone density studies. They listed diagnosis narratives, but failed to provide the corresponding ICD-9 CM codes. The office staff has attempted to ICD-9 CM code these conditions — with limited success. Can you assist us in properly coding the diagnoses listed?

Answer

You are obviously referring to Blue Shield's October 1996 *Bulletin*. This policy clarifies that a bone density study will be a covered service and medically necessary (unless excluded by a particular employer contract) for the specific conditions/diseases listed. While Blue Shield's diagnostic statement failed to match an ICD-9 CM code or a specific combination of ICD-9 CM codes, we have suggested diagnoses that are as close as possible to the diagnostic statement.

- **Hyperparathyroidism (can cause secondary osteoporosis): ICD-9 CM code 252.0**
(588.8 refers to secondary hyperparathyroidism of renal origin. Blue Shield did not specify the type of hyperparathyroidism and it would seem appropriate for Blue Shield to include it in its coverage.)
- **Hyperthyroidism (can cause secondary osteoporosis): ICD-9 CM code 242.9X**
Note that this ICD-9 CM code requires a 5th digit (0,1) to identify if there is a thyrotoxic crisis or not. We selected 242.9X because the hyperthyroidism specified in Blue Shield's *Bulletin* did not mention the presence of a nodular goiter. However, it would seem appropriate for Blue Shield to recognize the other hyperthyroidism ICD-9 CM codes (242.0X, 242.1X, 242.2X, 242.3X)
- **Cushing's syndrome (can cause secondary osteoporosis): ICD-9 CM code 255.0**
This is a syndrome that results from the hypersecretion of the adrenal cortex resulting in the excessive production of glucosteroids and characterized by specific conditions such as osteoporosis.
- **Long-term corticosteroid therapy (greater than one month) (can cause secondary osteoporosis): V58.69, 733.09 and E932.0**
An ICD-9 CM code that may describe this situation is V58.69 ("an encounter has occurred due to long-term and current use of medications."). Should the long-term use of the corticosteroid therapy cause osteoporosis then this condition would be a drug-induced osteoporosis 733.09 and E932.0 — Adrenal Cortical Steroids. ICD-9 CM instructions are to code the appropriate "E"

code to identify the drug. (Remember that an "E" ICD-9 CM code can never be the primary diagnosis code.)

- **Osteoporosis treated by specific medication (i.e., calcitonin): 733.00 (Osteoporosis unspecified) 275.4 (possibly; see explanation)**

Calcitonin is a hormone that is produced by the thyroid gland. It controls the level of calcium in the blood by slowing the rate at which calcium is lost from bones. Blue Shield allows one (1) bone mass measurement each year when a patient has already been diagnosed to have osteoporosis and synthetic calcitonin has been used to retard the bone resorption (loss). Since calcitonin decreases the calcium blood level, Blue Shield may recognize the diagnosis of hypercalcemia (275.4) to substantiate the prescription of calcitonin. Note: no specific ICD-9 CM code could be identified to specify drug therapy to reduce bone loss. You may find that a brief narrative may be necessary to adequately reflect this condition.

- **Risk for osteoporosis decides whether estrogen replacement therapy should be prescribed or not:**

This diagnostic narrative indicates that the patient has significant risk factor(s) for osteoporosis (i.e., disease such as above on medication such as gonadotropin releasing hormone agent (GnRH) DHIA) and would benefit from the prescription of estrogen. Such reasons or conditions that may fall into this category: premature menopause (256.3), family (mother) history of osteoporosis (V17.8). (After menopause the ovaries no longer produce estrogen. Estrogen helps to maintain bone mass. When a woman goes through menopause early, or if the mother had osteoporosis, then the female has increased risk of developing osteoporosis.)

Note: Blue Shield's coverage does not include the routine screening, diagnosis, and monitoring of osteoporosis. Blue Shield also will not cover the bone scan for patients already on estrogen therapy to treat menopause or for individuals where estrogen therapy is contraindicated.

It is always important to monitor your Blue Shield Explanation of Benefits to identify when Blue Shield has found the reported medical condition (diagnosis) did not appropriately describe or satisfy their established medical necessity. If you believe that your diagnosis code did relate to Blue Shield's medical necessity criteria, then you should request an appeal regarding the claim's adjudication. Include in your appeal how your diagnosis coding related to Blue Shield's specific guidelines.

DEBORAH BELL, CPC

Ms. Bell is a regional coding and reimbursement consultant certified in medical coding by the American Academy of Procedural Coders. ■

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

MGA/IMGs series, Department of Mental Hygiene, The Johns Hopkins University, School of Hygiene and Public Health, Keswick Nursing Center, 40th Street, 6:00 p.m. -7:00 p.m., light refreshments served at 5:30 p.m. Credits: TBA. Sponsored by the Maryland Chapter of the American Geriatrics Society (AGS) and the Maryland Gerontological Association (MGA), with assistance of the Geriatrics Committee of the Maryland Academy of Family Physicians. Info: Donna Meisel Weinreich, 410-675-3244 (e-mail: dmeisel@umabnet.ab.umd.edu) or Joseph J. Gallo, M.D., M.P.H., 410-955-0599 (e-mail: jgallo@welchlink.wlech.jhu.edu).

TB prevention in long-term care	Feb. 18
Perioperative management of the elder adult	Mar. 11
Pain management in the older adult	Apr. 29
Computed body tomography 1997: the cutting edge , sponsored by the Johns Hopkins Medical Institutions, Department of Radiology, at Peabody Orlando Hotel, Orlando, Florida. Comprehensive review of recent advances in computed body tomography with some correlation with MRI. Credits: 21 Cat 1 AMA credits; 21 Cat A CE credits as designated by the ASRT, CEUs from the Florida HRS Office of Radiation Control. Fee: \$575/physicians; \$500/residents, fellows, technologists.	Feb. 6-9
14th annual gastroenterology update: multi disciplinary approach — an exercise in interactive gastroenterology , Silvertree Hotel, Snowmass Village, Aspen, Colorado. Credits: 19 Cat 1 AMA credits. Fee: \$495/physicians; \$375/residents, fellows, allied health professionals.	Feb. 9-14
Perioperative management: a course designed for practitioners to limit patient risk by proper pre- and postoperative evaluation and care , Marriott's Marco Island Resort and Golf Club, Marco Island, Florida. Credits: 20.5 Cat 1 AMA credits; 20 CE credits by the AANA. Fee: \$525/physicians (After Feb. 9, 1997: \$550); \$490/residents, fellows, CRNs (After Feb. 9, 1997: \$515).	Mar. 9-12
Management of auditory and vestibular disorders and vestibular practicum . Credit: TBA. Fee: \$400/physicians; \$300/residents, fellows, allied health professionals. Practicum: \$75.	Mar. 12-13
What's new in the "memory wars": implications for clinical practice , sponsored by the Department of Psychiatry and Behavioral Sciences and False Memory Syndrome Foundation. Credits: 8 Cat 1 AMA credits. Fee: \$175.	Mar. 21
Second annual cardiovascular symposium with the experts , sponsored by the Department of Medicine at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 11.5 Cat 1 AMA credits. Fee: \$175 by March 30, \$225 after March 30.	Mar. 30-31
Diagnosis and treatment of neoplastic disorders , sponsored by The Johns Hopkins Oncology Center. Credits: 14 Cat 1 AMA credits. Fee: \$300/Advanced registration (before Feb. 1); \$325 (Postmarked Feb. 1 and after); \$150/residents, fellows, allied health professionals.	Apr. 3-4
Nuclear oncology: from genotype to patient care , sponsored by The Johns Hopkins University School of Medicine. Credits: up to 18 Cat 1 AMA credits. Fee: \$495/physicians; \$395/residents, fellows, allied health professionals.	Apr. 7-9
Update on Alzheimer's disease and other dementias , Renaissance Harborplace Hotel, Baltimore, MD. Credits: up to 7 Cat 1 AMA credits. Fee: \$145/physicians; \$110/psychologists in practice; \$90/residents, fellows, allied health professionals.	Apr. 12
25th annual pediatric trends , Johns Hopkins Medical Institute, Department of Pediatrics. This course provides a comprehensive update on new developments of interest to practitioners who care for infants, children, and adolescents. Credits: 42.5 Cat 1 AMA credits; 45.5 AAP credits; 37.5 AAFP prescribed hours.	Apr. 14-19

The Johns Hopkins Medical Institutions (continued)

- 38th annual postgraduate institute for pathologists in clinical cytopathology**, sponsored by The Johns Hopkins University School of Medicine. Credits: 94.5 Cat 1 AMA credits plus up to 10 hrs. video instruction. Fee: \$2450/physicians; \$1300/senior residents.
- Course A** (Home study) Mar.–Apr.
- Course B**, concentrated lecture and laboratory studies, Johns Hopkins Medical Institutions, Baltimore, MD. Apr. 14–25
- Seventh annual clinical care of the patient with HIV infection**, sponsored by the Department of Medicine at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$190/residents, fellows, allied health professionals. Apr. 17–18
- 11th annual mood disorders symposium**, sponsored by The Johns Hopkins Affective Disorders Clinic, and DRADA. Credit: Cat 1 AMA credit; Cat A credit, Md. State Board of Examiners of Psychologists; Md. State Board of Examiners for Social Workers. Fee: \$50/DRADA members; \$60/other attendees. Apr. 30
- Institute on ministry with the sick**, sponsored by the Johns Hopkins University School of Medicine. Credits: 14 Cat 1 AMA credits. Fee: \$150. May 5–7
- Pediatric allergy and immunology for the practitioner**, sponsored by the Division of Pediatric Allergy and Immunology. Credits: up to 14 Cat 1 AMA credits. Fee: If postmarked by April 1, \$275/physicians; \$200/residents, other allied health professionals. If postmarked after April 1, \$295/physicians; \$220/residents, other allied health professionals. May 8–9
- Critical issues in surgical pathology**, sponsored by the Department of Pathology. Credits: 14 Cat 1 AMA credits. Fee: \$400/physicians; \$200/residents, fellows, students. May 9–10
- 42nd annual topics in clinical medicine**, sponsored by the Department of Medicine. Credits: 39 Cat 1 AMA credits. Fee: \$750/physicians; \$600/residents, fellows, other professionals. May 12–16

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956) or by calling the phone number listed after a specific program. Fax 410-706-3103.

- Diagnostic and therapeutic advances in glaucoma management**, Sheraton Inner Harbor, Baltimore, MD. Credits: TBA. Feb. 21

Self-Directed CME Activities

- Disease management of lipid disorders (audio tape and test).** Credit: 1 Cat 2 AMA credit. Expires 6/97.

University of Maryland School of Medicine (continued)

CD-ROM based interactive multimedia radiology teaching file for Mac or PC w/single user licenses (SUL), site licenses (SL), or multisite licenses (MSL). Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference, each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Office of Continuing Medical Education, Washington University School of Medicine, Campus Box 8063, 660 South Euclid Ave., St. Louis, MO 63110-1093. Unless otherwise noted, seminars will be held at the Washington University Medical Center, Eric P. Newman Education Center (EPNEC), 320 S. Euclid Ave., St. Louis, MO 63110. Info: Cathy Sweeney, 800-325-9862, Fax 314-362-1087.

Internal medicine review, Monday evenings, The Jewish Hospital.

Mar.–May

Integrated care of the thoracic surgery patient: a seminar for allied health professionals.

Mar. 21–22

Clinical pulmonary update.

Apr. 4–5

Refresher course & update in general surgery, The Ritz-Carlton Hotel, St. Louis, MO.

Apr. 10–12

Delmarva Foundation for Medical Care, Easton, MD. Credits: 3 Cat 1 AMA credits. Info: Roxanne Rodgers, 410-822-0697.

Health care improvement for physicians

Feb. 5

Health care improvement for physicians

Feb. 12

Health care improvement for physicians

Feb. 14

Health care improvement for physicians

Feb. 15

Health care improvement for physicians

Mar. 14

Health care improvement for physicians

Mar. 15

Medical care of women in the era of HIV disease (three-part series), The Medical and Chirurgical Faculty of Maryland, 1211 Cathedral St., Baltimore, MD, 7:00 a.m. - 9:00 a.m. Credits: maximum of 6 Cat 1 AMA credits available. Fee: \$15/session; \$40/series.

Clinical management of the HIV-positive woman

Feb. 5

Cardiovascular conference at Snowshoe, Mountain Lodge Conference Center, Snowshoe, West Virginia. Sponsor: American College of Cardiology (ACC). Credits: 14 Cat 1 AMA credits. Info: Registration Secretary, Extramural Programs Dept., ACC, 9111 Old Georgetown Rd., Bethesda, MD 20814-1699. 800-253-4636 ext. 695, Fax 301-897-9745.

Feb. 3–5

Magnetic resonance imaging of the brain, spine and musculoskeletal system, sponsored by the University of California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, California. Credits: 30 Cat 1 AMA credits. Fee: \$695/physicians; \$495/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

Mar. 2–7

Sports medicine for the clinician, Vail Cascade Hotel & Club, Vail, Colorado. Presented by Division of Family Medicine; Department of Orthopedic Surgery; and the Office of Continuing Medical Education, UCLA School of Medicine. Credits: up to 20 Cat 1 AMA credits. Fee: \$595/physicians; \$300/residents, other health care professionals. Contact: Office of CME, 310-794-2620.

Mar. 3–7

Management of the HIV-infected patient: a practical approach for the primary care practitioner, Crowne Plaza Manhattan, New York, NY. Sponsor: The Center for Bio-Medical Communication, Inc. (CBC) in collaboration with the American Foundation for AIDS Research. Credits: 20.25 Cat 1 AMA credits. Fee: Before Jan. 24, \$495/

Mar. 7–9

Miscellaneous (continued)

physicians; \$295/physicians-in-training and allied health professionals. Contact: CBC, 201-385-8080, Fax 201-385-5650 (e-mail: cbcbiomed@aol.com).

- 4th annual update in general diagnostic imaging: breast, abdominal and neuroradiology imaging**, sponsored by the University of Chicago, Department of Radiology, at The Breakers Resort Hotel, Palm Beach, Florida. Credits: 25 Cat 1 AMA credits (includes 15 hrs. in mammography). Fee: \$700/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Mar. 10-14**
- Minimally invasive therapy of the brain**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Hotel, Marina Del Rey, California. Credits: 17.75 Cat 1 AMA credits. Fee: \$395/physicians; \$300/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Mar. 14-16**
- Eastern wisdom and the practice of psychotherapy**, conference at The Conference Center at Sheppard Pratt, Baltimore, Maryland. Info: Barbara Johnson, Professional Education Programs, Sheppard Pratt Health System, 410-938-4598, (e-mail: riamy@capcon.net). **Mar. 22**
- 1997 annual session, American College of Physicians**, at the Pennsylvania Convention Center, Philadelphia. The largest meeting for internal medicine and its subspecialties. CME credit available. Pre-session courses March 20-21. Info: 800-523-1546, ext. 2600. **Mar. 22-25**
- NIH consensus development conference on management of hepatitis C**, Natcher Conference Center, National Institutes of Health, Bethesda, Maryland. Credits: up to 15 Cat 1 AMA credits. Fee: none; early registration encouraged. Contact: Rose Salton, 301-770-3153, Fax 301-468-2245 (e-mail: confdept@tech-res.com). **Mar. 24-26**
- Problem solving in diagnostic radiology**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Palm Beach, Florida. Credits: 30 Cat 1 AMA credits. Fee: \$694/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Mar. 29**
- 17th annual resident's radiology review course**, sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, California. Designed for senior radiology residents and practicing radiologists. Course covers all major modalities. Credits: 41 Cat 1 AMA credits. Fee: \$695/physicians; \$450/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-8959, (e-mail: webmaster@ryalsmeet.com). **Mar. 30-Apr. 4**
- Reimbursement and managed care: essential reimbursement strategies in emergency medicine**, Hyatt Regency, Baltimore, Maryland. Sponsor: the American College of Emergency Physicians (ACEP). Credits: 15 Cat 1 AMA credits; 15 Cat 1 ACEP credits. Info: 800-798-1822. **Apr. 3-4**
- Breast imaging and interventions**, sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, California. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$250/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 4-6**
- Getting control: effective procedure coding for emergency medicine**, Hyatt Regency, Baltimore, Maryland. Sponsor: the American College of Emergency Physicians (ACEP). Credits: 15.5 Cat 1 AMA credits; 15.5 Cat 1 ACEP credits. Info: 800-798-1822. **Apr. 4-6**
- Leadership conference**, Stouffer Mayflower Hotel, Washington, DC. Sponsor: American College of Emergency Physicians (ACEP). Credits: TBA. Info: 800-798-1822. **Apr. 6-7**

Miscellaneous (continued)

- Legislative issues forum**, Stouffer Mayflower Hotel, Washington, DC. Sponsor: the American College of Emergency Physicians (ACEP). Credits: TBA. Info: 800-798-1822. **Apr. 7-9**
- Building a multidiscipline team for the diagnosis and management of breast disease**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Plaza Hotel, New York, NY. Credits: 21 Cat 1 AMA credits. Fee: \$595/hospital team (no fee for every fifth member — must register together to qualify). Info: Ryals & Associates, 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 10-13**
- Infectious disease '97: a comprehensive review for the practicing physician**, sponsored by The Center for Bio-Medical Communication, Inc. (CBC), at Renaissance Washington, D.C. Hotel. Credits: 18.25 Cat 1 AMA credits; 18.25 AAFP credits. Fee: by Feb. 3, 1997, \$495/physicians; \$350/physicians-in-training, other allied health professionals. Info: 201-385-8080, Fax 201-385-5650 (e-mail: webmaster@ryalsmeet.com). **Apr. 11-13**
- Sixth annual meeting and clinical congress of the American Association of Clinical Endocrinologists (AACE)**, Marriott, Philadelphia, Pennsylvania. Credits: up to 36.5 Cat 1 AMA credits. Info: 904-353-7878. **Apr. 16-20**
- Problem solving in imaging of the brain, spine, and head and neck**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Amelia Island, Florida. There will be both didactic lectures and workshops. Credits: 26 Cat 1 AMA credits. Fee: \$650/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 17-20**
- 2nd annual angio/interventional review course**, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Credits: 10 Cat 1 AMA credits. Fee: \$215/physicians; \$155/residents, fellows, full-time military, U of F radiology alumni (\$135 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 19-20**
- 9th annual radiology review course: "what you need to know,"** sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Attendees will improve their knowledge of differential diagnosis, imaging patterns, and techniques of examination. Credits: 50 Cat 1 AMA credits. Fee: \$695/physicians; \$525/residents, fellows, full-time military, U of F radiology alumni, (\$475 each two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 701-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 20-25**
- Critical care medicine '97: 11th annual review and update**, sponsored by the Center for Bio-Medical Communication, Inc., Hyatt Regency, Washington, D.C. Credits: 41.25 Cat 1 AMA credits, 41.25 AAFP. Fee: By Mar. 21, 1997, \$795/physicians; \$575/physicians-in-training, allied professionals. Info: 201-385-8080, Fax 201-385-5640 (e-mail: cbcbiomed@aol.com). **Apr. 30-May 4**
- 2nd annual mammography review course**, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Course is designed as an overview of the practical aspects of breast imaging, including interventional procedures. Credits: 15 Cat 1 AMA credits. Fee: \$295/physicians; \$215/residents, fellows, full-time military, U of F Radiology Alumni (\$185 each, two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 25-27**

Miscellaneous (continued)

- Clinical auscultation of the heart**, Georgetown University Medical Center, Washington, D.C. **May 7-9**
Sponsored by the American College of Cardiology. Credits: 21 Cat 1 AMA credits.
Contact: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, MD 20814-1699, 800-253-4636, ext. 695, Fax 301-897-9745.
- 56th annual American occupational health conference: discover the reality**, Orange County Convention Center, Orlando, Florida. **May 9-16**
39 concurrent scientific sessions, 42 postgraduate seminars, and 7 two-day training courses. Sponsors: the American College of Occupational and Environmental Medicine (ACOEM) in conjunction with American Occupational Health Conference (AOHC). Contact: Kay Cone, ACOEM, 55 W. Seegers Rd., Arlington Heights, IL 60005, 847-228-6850, ext. 152, Fax 847-228-1856.
- Cutaneous melanoma '96: a clinical symposium for primary care practitioners**, sponsored by The Skin Cancer Foundation and Memorial Sloan-Kettering Cancer Center, Memorial Sloan-Kettering Cancer Center, New York, NY. **May 16**
Credits: 6.5 Cat 1 AMA credits.
Info: Ludmilla Popoff, 212-639-6754.
- 2nd Annual mammography — practical challenges of the '90's**, sponsored by X-Ray Associates of New Mexico, P.C., at The Eldorado Hotel, Sante Fe, New Mexico. **May 23-26**
Credits: 20 Cat 1 AMA credits. Fee: \$650/physicians; \$450/residents, fellows; \$350/technologists, nurses. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

Self-Directed CME Activities

- Maryland physicians' campaign against family violence, module one: domestic violence**, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.
- Maryland physicians' campaign against family violence, module two: child maltreatment**, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

- Fluorescein angiography conference**, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00 a.m. – 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.
- Sinai Hospital of Baltimore medical grand rounds**, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.



EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

February, 1997

Foodborne Disease Outbreaks in Maryland

In Maryland, confirmed or suspected disease outbreaks, including foodborne outbreaks, are reportable by health care providers to their local health department under Code of Maryland Regulations 10.06.01. Foodborne outbreaks account for approximately one third of all outbreaks each year (Figure 1). On the average, 63 foodborne outbreaks are reported each year; Figure 2 portrays the number of foodborne outbreaks by year from 1986 to 1995. In the majority of foodborne outbreaks reported in 1993-1995, the etiologic agents have been unknown (71%). *Salmonella*, however, has been responsible for 37 (55%) of the 67 foodborne outbreaks with known etiology for that period. The remaining 29% with known etiologies were due to fish poisoning (scombroid,

ciguatera, paralytic fish poisoning) (13), Norwalk or Norwalk-like virus (5), *Bacillus cereus* (2), hepatitis A (2), strep throat (2), and one each of campylobacteriosis, *Clostridium perfringens*, *E. coli* hemolytic uremic syndrome, giardiasis, shigellosis, and trichinosis.

The following table provided by the Centers for Disease Control and Prevention (CDC) is intended for use as a guideline for confirming foodborne disease outbreaks due to selected etiologic agents [Morbidity and Mortality Weekly Report (MMWR), October 25, 1996, Vol. 5, No. SS-5]. Information is provided on incubation periods, clinical syndromes, and criteria for confirming the etiologic agent.

Figure 1. Outbreaks Reported in Maryland, 1993-1995, by Mode of Transmission

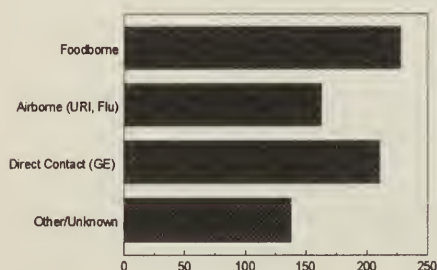
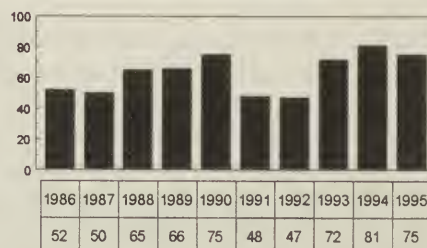


Figure 2. Foodborne Outbreaks Reported in Maryland, 1986-1995



Guidelines for Confirmation of Foodborne-Disease Outbreaks

[Morbidity and Mortality Weekly Report (MMWR), October 25, 1969, Vol. 5, No. SS-5]

<u>Etiologic Agent</u>	<u>Incubation Period</u>	<u>Clinical Syndrome</u>	<u>Confirmation</u>
Bacterial			
1. <i>Bacillus cereus</i>			
a. Vomiting toxin	1-6 hrs	Vomiting, some patients with diarrhea; fever uncommon	Isolation of organism from stool of two or more ill persons and not from stool of controls OR Isolation of $\geq 10^5$ organisms/g from epidemiologically implicated food, provided specimen properly handled
b. Diarrheal toxin	6-24 hrs	Diarrhea, abdominal cramps, and vomiting in some patients; fever uncommon	Isolation of organism from stool of two or more ill persons and not from stool of controls OR Isolation of $\geq 10^5$ organisms/g from epidemiologically implicated food, provided specimen properly handled
2. <i>Brucella</i>	Several days to several mos, usually >30 days	Weakness, fever, headache, sweats, chills, arthralgia, weight loss, splenomegaly	Two or more ill persons and isolation or organism in culture of blood or bone marrow, greater than fourfold increase in standard agglutination titer (SAT) over several wks, or single SAT titer $\geq 1:160$ in person who has compatible clinical symptoms and history of exposure
3. <i>Campylobacter</i>	2-10 days, usually 2-5 days	Diarrhea (often bloody), abdominal pain, fever	Isolation of organism from clinical specimens from two or more ill persons OR Isolation of organism from epidemiologically implicated food
4. <i>Clostridium botulinum</i>	2 hrs-8 days, usually 12-48 hrs	Illness of variable severity; common symptoms are diplopia, blurred vision, and bulbar weakness; paralysis, which is usually descending and bilateral, may progress rapidly	Detection of botulinum toxin in serum, stool, gastric contents, or implicated food OR Isolation of organism from stool or intestine

5. <i>Clostridium perfringens</i>	6-24 hrs	Diarrhea, abdominal cramps; vomiting and fever are uncommon	Isolation of $\geq 10^6$ organisms/g in stool of two or more ill persons, provided specimen properly handled OR Demonstration of enterotoxin in the stool of two or more ill persons OR Isolation of $\geq 10^5$ organisms/g from epidemiologically implicated food, provided specimen properly handled
6. <i>Escherichia coli</i>			
a. Enterohemorrhagic (<i>E. coli</i> O157:H7 and others)	1-10 days, usually 3-4 days	Diarrhea (often bloody), abdominal cramps (often severe), little or no fever	Isolation of <i>E. coli</i> O157:H7 or other Shiga-like toxin-producing <i>E. coli</i> from clinical specimen of two or more ill persons OR Isolation of <i>E. coli</i> O157 or other Shiga-like toxin-producing <i>E. coli</i> from epidemiologically implicated food
b. Enterotoxigenic (ETEC)	6-48 hrs	Diarrhea, abdominal cramps, nausea; vomiting and fever are less common	Isolation of organism of same serotype, which are demonstrated to produce heat-stable (ST) and/or heat-labile (LT) enterotoxin, from stool of two or more ill persons
c. Enteropathogenic (EPEC)	Variable	Diarrhea, fever, abdominal cramps	Isolation of same enteropathogenic serotype from stool of two or more ill persons
d. Enteroinvasive (EIEC)	Variable	Diarrhea (may be bloody), fever, abdominal cramps	Isolation of same enteroinvasive serotype from stool of two or more ill persons
7. <i>Listeria monocytogenes</i>			
a. Invasive disease	2-6 wks	Meningitis, neonatal sepsis, fever	Isolation of organism from normally sterile site
b. Diarrheal disease	Unknown	Diarrhea, abdominal cramps, fever	Isolation of organism of same serotype from stool of two or more ill persons exposed to food that is epidemiologically implicated or from which organism of same serotype has been isolated

- | | | | |
|-----------------------------------|---------------------------------------|--|---|
| 8. Nontyphoidal <i>Salmonella</i> | 6 hrs-10 days,
usually 6-48
hrs | Diarrhea, often with fever and
abdominal cramps | Isolation of organism of same serotype from clinical specimens
from two or more ill persons

OR
Isolation of organism from epidemiologically implicated food |
| 9. <i>Salmonella typhi</i> | 3-60 days,
usually 7-14
days | Fever, anorexia, malaise,
headache, and myalgia;
sometimes diarrhea or
constipation | Isolation of organism from clinical specimens of two or more ill
persons

OR
Isolation of organism from epidemiologically implicated food |
| 10. <i>Shigella</i> | 12 hrs-6 days,
usually 2-4
days | Diarrhea (often bloody),
frequently accompanied by
fever and abdominal cramps | Isolation of organism of same serotype from clinical specimens
from two or more ill persons

OR
Isolation of organism from epidemiologically implicated food |
| 11. <i>Staphylococcus aureus</i> | 30 min-8 hrs,
usually 2-4 hrs | Vomiting, diarrhea | Isolation of organism of same phage type from stool or vomitus of
two or more ill persons

OR
Detection of enterotoxin in epidemiologically implicated food

OR
Isolation of $\geq 10^5$ organisms/g from epidemiologically implicated
food, provided specimen properly handled |
| 12. <i>Streptococcus</i> Group A | 1-4 days | Fever, pharyngitis, scarlet
fever, upper respiratory
infection | Isolation of organism of same M- or T-type from throats of two or
more ill persons

OR
Isolation of organism of same M- or T-type from epidemiologically
implicated food |

13. *Vibrio cholerae*
a. O1 or O139
- 1-5 days
- Watery diarrhea, often accompanied by vomiting
- Isolation of toxigenic organism from stool or vomitus of two or more ill persons
OR
Significant rise in vibriocidal, bacterial-agglutinating, or antitoxin antibodies in acute- and early convalescent-phase sera among persons not recently immunized
OR
Isolation of toxigenic organism from epidemiologically implicated food
OR
Isolation of organism of same serotype from stool of two or more ill persons
OR
Isolation of Kanagawa-positive organism from stool of two or more ill persons
OR
Isolation of $\geq 10^5$ Kanagawa-positive organisms/g from epidemiologically implicated food, provided specimen properly handled
OR
Isolation of organism from clinical specimen of two or more ill persons
OR
Isolation of pathogenic strain of organism from epidemiologically implicated food
- b. non-O1 and non-O139
- 1-5 days
- Watery diarrhea
14. *Vibrio parahaemolyticus*
- 4-30 hrs
- Diarrhea
15. *Yersinia enterocolitica*
- 1-10 days, usually 4-6 days
- Diarrhea, abdominal pain (often severe)

Chemical

1. Marine toxins
- a. Ciguatera
- 1-48 hrs, usually 2-8 hrs
- Usually gastrointestinal symptoms followed by neurologic symptoms (including paresthesia of lips, tongue, throat, or extremities) and reversal of hot and cold sensation
- Demonstration of ciguatera toxin in epidemiologically implicated fish
OR
Clinical syndrome among persons who have eaten a type of fish previously associated with ciguatera fish poisoning (e.g., snapper, grouper, or barracuda)

b. Scombroid toxin (histamine)	1 min-3 hrs, usually <1 hr	Flushing, dizziness, burning of mouth and throat, headache, gastrointestinal symptoms, urticaria, and generalized pruritus	Demonstration of histamine in epidemiologically implicated food OR Clinical syndrome among persons who have eaten type of fish previously associated with histamine fish poisoning (e.g., mahi- mahi or fish of order Scombroidei)
c. Paralytic or neurotoxic shellfish poison	30 min-3 hrs	Paresthesia of lips, mouth or face, and extremities; intestinal symptoms or weakness, including respiratory difficulty	Detection of toxin in epidemiologically implicated food OR Detection of large numbers of shellfish-poisoning-associated species of dinoflagellates in water from which epidemiologically implicated mollusks are gathered
d. Puffer fish, tetrodotoxin	10 min-3 hrs, usually 10-45 mins	Paresthesia of lips, tongue, face, or extremities, often following numbness; loss of proprioception or "floating" sensations	Demonstration of tetrodotoxin in epidemiologically implicated fish OR Clinical syndrome among persons who have eaten puffer fish
2. Heavy metals a. Antimony b. Cadmium c. Copper d. Iron e. Tin f. Zinc	5 min-8 hrs, usually <1 hr	Vomiting, often metallic taste	Demonstration of high concentration of metal in epidemiologically implicated food
3. Monosodium glutamate (MSG)	3 min-2 hrs, usually <1 hr	Burning sensation in chest, neck, abdomen, or extremities; sensation of lightness and pressure over face or heavy feeling in chest	Clinical syndrome among persons who have eaten food containing MSG (i.e., usually ≥ 1.5 g MSG)

4. Mushroom toxins
 - a. Shorter-acting toxins: ≤ 2 hrs

Muscimol
Muscarine
Psilocybin
Coprinus atramentarius
Ibotenic acid

Usually vomiting and diarrhea, other symptoms differ with toxin:
 Confusion, visual disturbance
 Salivation, diaphoresis
 Hallucinations
 Disulfiram-like reaction
 Confusion, visual disturbance
 - b. Longer-acting toxin (e.g., *Amanita* spp.)

6-24 hrs

Diarrhea and abdominal cramps for 24 hrs followed by hepatic and renal failure

OR

Demonstration of toxin in epidemiologically implicated mushroom or mushroom-containing food

Parasitic

1. *Cryptosporidium parvum*

2-28 days, median: 7 days

Diarrhea, nausea, vomiting, fever

Demonstration of organism or antigen in stool or in small-bowel biopsy of two or more ill persons

OR

Demonstration of organism in epidemiologically implicated food
2. *Cyclospora cayentanensis*

1-11 days, median: 7 days

Fatigue, protracted diarrhea, often relapsing

Demonstration of organism in stool of two or more ill persons
3. *Giardia lamblia*

3-25 days, median: 7 days

Diarrhea, gas, cramps, nausea, fatigue

Two or more ill persons and detection of antigen in stool; or demonstration of organism in stool, duodenal contents, or small-bowel biopsy specimen
4. *Trichinella* spp.

1-2 days for intestinal phase; 2-4 wks for systemic phase

Fever, myalgia, periorbital edema, high eosinophil count

Two or more ill persons and positive serologic test or demonstration of larvae in muscle biopsy

OR

Demonstration of larvae in epidemiologically implicated meat

Viral

1. Hepatitis A

15-50 days,
median: 28
days

Jaundice, dark urine, fatigue,
anorexia, nausea

Detection of IgM anti-hepatitis A virus in serum from two or more persons who consumed epidemiologically implicated food
2. Norwalk family of viruses,
small round-structured
viruses (SRSV)

15-77 hrs,
usually 24-48
hrs

Vomiting, cramps, diarrhea,
headache

More than fourfold rise in antibody titer to Norwalk virus or Norwalk-like virus in acute and convalescent sera in most serum pairs

OR

Visualization of small, round-structured viruses that react with patient's convalescent sera but not acute sera-by immune-electron microscopy. Assays based on molecular diagnostic (e.g., polymerase-chain reaction [PCR], probes, or assays for antigen and antibodies from expressed antigen) are available in reference laboratories.
3. Astrovirus, calicivirus,
others

15-77 hrs,
usually 24-48
hrs

Vomiting, cramps, diarrhea,
headache

Visualization of small, round-structured viruses that react with patient's convalescent sera but not acute sera-by immune-electron microscopy. Assays based on molecular diagnostics (e.g., PCR, probes, or assays for antigen and antibodies from expressed antigen) are available in reference laboratories.

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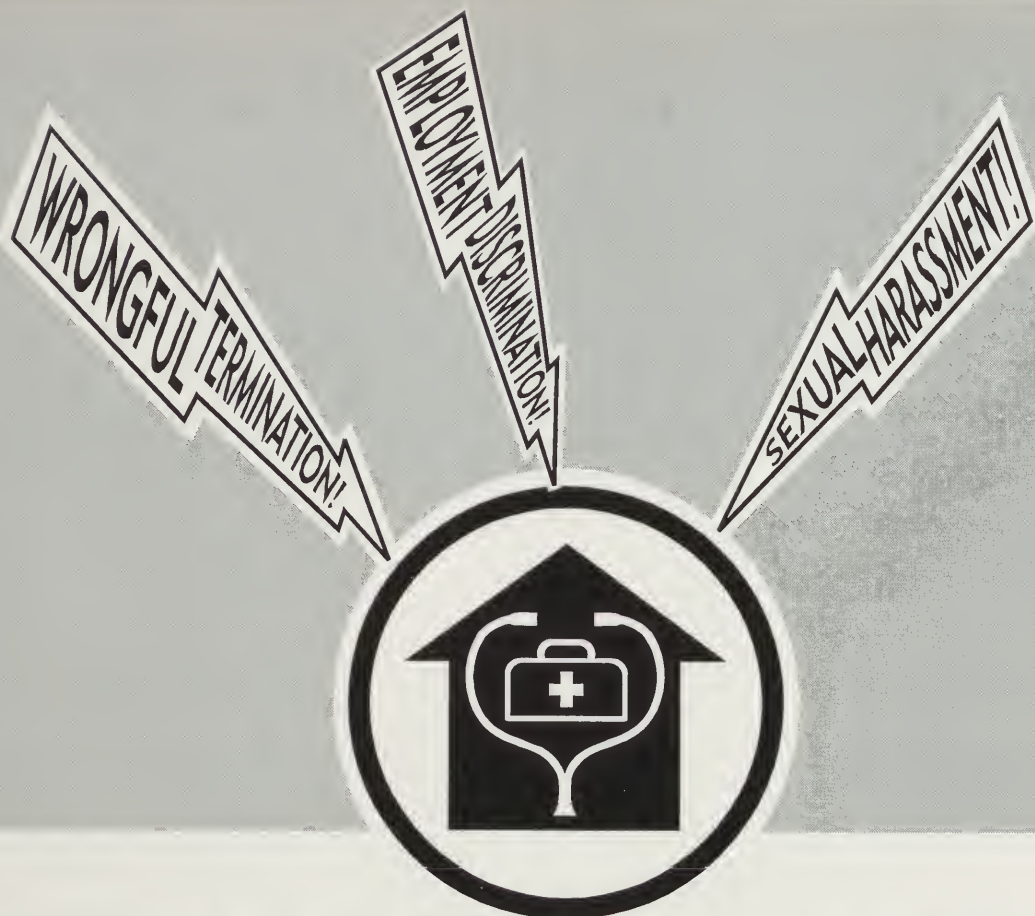
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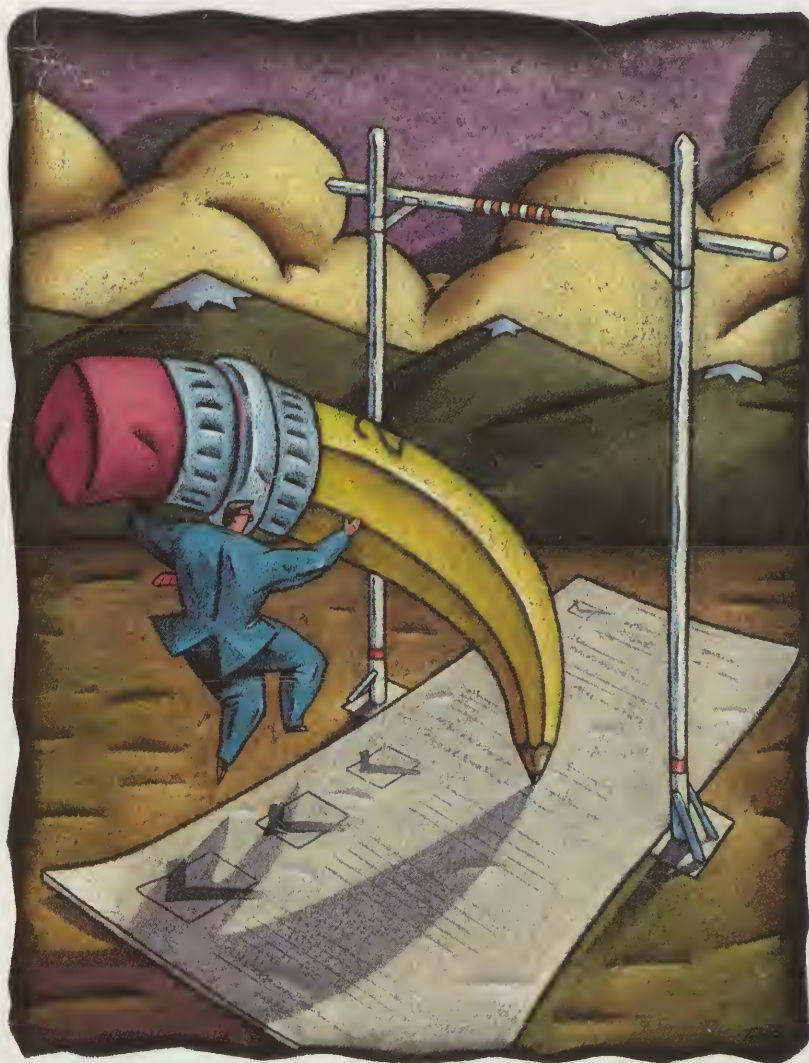
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Original Articles

*Regional/local anesthesia: a safe
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undergoing carotid endarterectomy*
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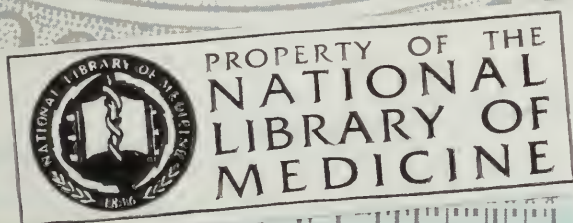
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Extensive pneumatosis intestinalis
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*Oncology Today
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—141—

MARCH 1997



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The Gamma Knife

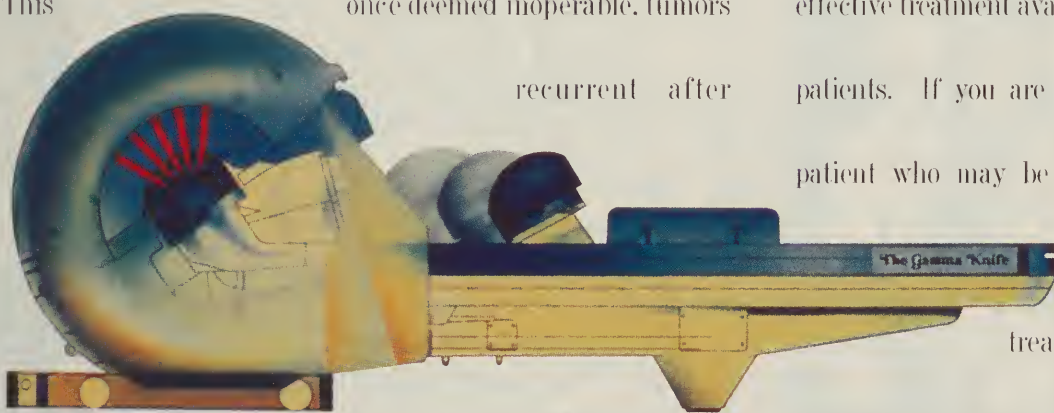
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


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Roy A.M. Meyers, M.D.,
winner, 1996 Med Chi
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Seventeenth Annual Med Chi Photo Contest

Rules:

-  Open to Med Chi and Alliance members only. Professional photographers may not enter.
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-  Photographs must measure at least 8" x 10" but be no larger than 11" x 14".
-  Entries must be matted or dry mounted. Framed photographs will not be accepted.
-  Name, address and telephone number of entrant must be on back of each entry.
-  Entries may be forwarded to: Med Chi, 1211 Cathedral Street, Baltimore, MD 21201,
Attn: Photo Contest no later than 5:00 p.m., Friday, April 18, 1997. (Limit 3 entries per person.)
-  All entries will be on display at the Turf Valley Inn and Convention Center, Ellicott City during the meeting.
-  First, second and third place winners will be announced May 2, 1997 at the annual meeting of the Medical & Chirurgical Faculty.
-  Photographs may be claimed at the registration desk at the close of the annual meeting May 3, or at Med Chi offices thereafter.

Med Chi is not responsible for loss of or damage to photographs submitted to contest.



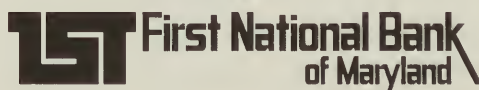


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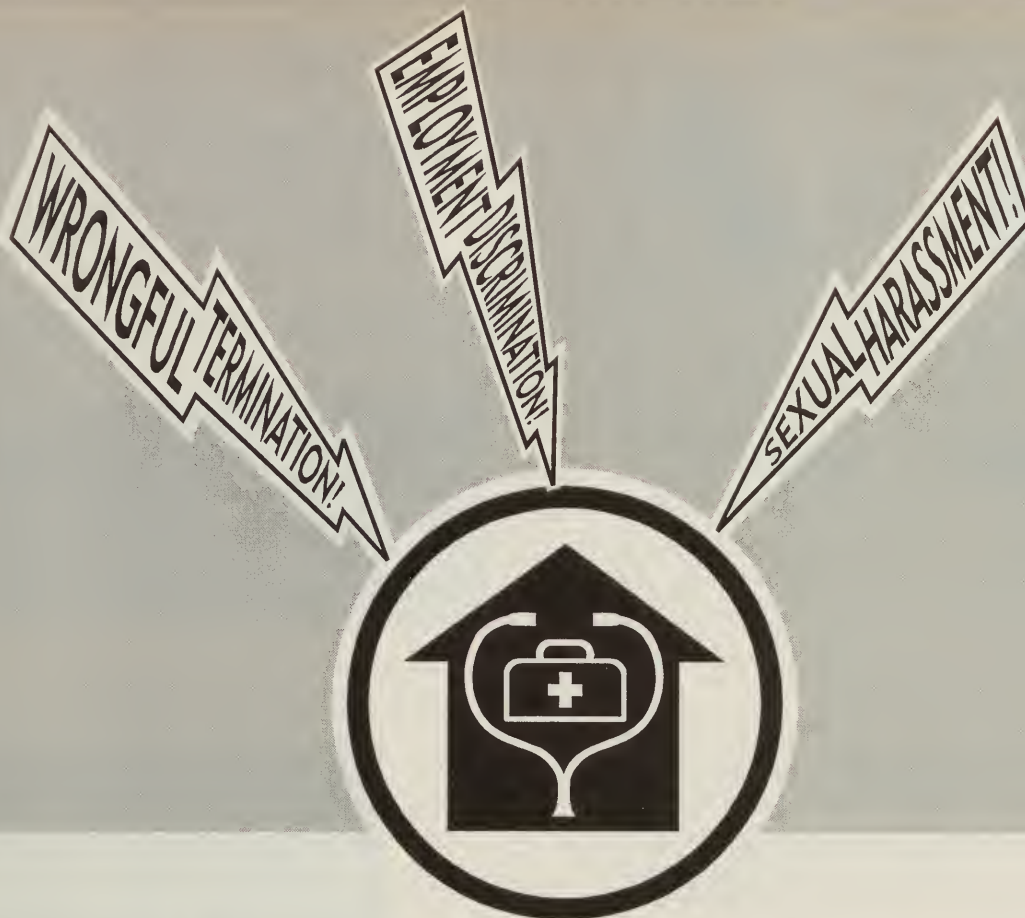
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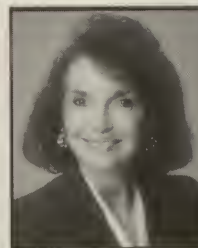
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What happened to the *MMJ*?

Will someone please explain what happened at the *Maryland Medical Journal*? I got into a routine at the first of the month, anticipating a pleasant read of your magazine: a few features, an article or two, and some local interest. The content wasn't world class, but it wasn't too shabby either. Even the covers were pleasant; no more politics and promotions, just appropriate designs. The scenes of Maryland photographed by Med Chi members were particularly attractive. Some of your special issues on smoking, sports medicine, and World War II had some great stuff—better than national journal articles.

I guess I got lulled into acceptance. The trend seemed positive. With new authors and clinical series it looked as if Med Chi

and the medical schools were getting on the same band wagon. So when did the wheels come off?

I took a close look—new cover, new paper, new surveys (where are the results?), and new masthead. Since the beginning of 1996, the managing editor, editor, associate editor, staff advisor, production assistant, and communications director have all disappeared. Is there an emerging virus on Cathedral Street? Have these people been purged? Do they live on in the Gulag?

Don't get me wrong. I still read the journal and appreciate your efforts, but can anybody tell me — what is the plan?

A. KERR MUDGEON, M.D. ■

Changes revive journal's scientific focus

Editor responds

Thank you for your letter. The editorial board welcomes comments, pro and con, concerning its efforts. Only in this fashion can we produce a journal which you will wish to read.

The changes you noted seeing in the January issue were directed by Med Chi's Board of Trustees, not the editorial board. We were advised that they feel the membership would be better served by separating "the legislative, regulatory, and medical society news" from its "scientific purposes." They indicated that in their view you would thus receive certain business information critical to your needs in a more timely manner. Alex Azar, M.D., Med Chi president and Michael Preston, acting executive director, commented on these anticipated changes in the December issue (pages 969 and 970) and Dr.

Azar did so again in the January issue (page 5).

The changes in personnel to which you refer were primarily for personal reasons and are best directed to those involved.

Your editor and editorial board who are primarily responsible for the contents of the *Maryland Medical Journal* wish to continue their charge by planning scientific and related presentations and hope to constantly improve the quality. Any comments which will aid us in this goal would be appreciated. Those which fall outside of our purview are better directed to the members of your board of trustees, charged with the overall direction of Med Chi.

MARION FRIEDMAN, M.D.
Editor ■

Use common sense as counsel

I regretted learning of the humiliation to which a colleague was subjected in a recent lawsuit. Although the court ultimately declared the physician not guilty, the aura of the catastrophe will linger.

When I started the practice of general surgery, my malpractice premium was \$90 per year. I was recently told that should I apply for the same privileges now, coverage would be about \$24,000. Exposure to the numerous cases of legal action has produced an explosive atmosphere.

Prudence suggests that we circle the wagons and approach this particular solvable problem. Although we live in an increasingly litigious society, we can protect ourselves from one specific onslaught by a simple action.

A law mandating the presence of a chaperon does not exist. Med Chi, appearing as an *amicus curiae* in the case referenced above, submitted a brief to the court asserting that "it was not always necessary or appropriate to have a chaperon present during a physical examination."

Common sense and self-protection counsel that these situations may be avoided entirely by having a chaperon present when any type of physical examination is done on a patient. The cost to the doctor may be slightly enhanced, but the removal of a possible lawsuit and its horrible sequelae are well worth the additional operating expense.

JOSEPH M. MILLER, M.D.

Dr. Miller is a retired surgeon in Timonium, Maryland. ■

Time to take individual action

While the jury is apparently out on whether managed care is bad for the nation's overall health,¹ few who have been providers within the traditional health care/indemnity model would be so nonjudgmental. Most of these providers believe the quality of care has suffered whether or not the aggregate statistics on morbidity and mortality would bear them out. Certainly there would be near unanimity that providers' quality of life has deteriorated. Let us not pass this off as an unimportant off-shoot of change; enjoyment of one's role as a provider does have repercussions on the quality of our work. We know this intuitively. It applies to the assembly line worker and it certainly applies to a practitioner of the healing arts.

The response to the seismic changes in health care has been evident in organized medicine's pursuit of both legislative² and judicial remedies. But I want to urge individual physicians to consider a role each may play in the battle against the erosion of quality health care when they witness their patients' care deteriorating because of the intrusion of managed care oversight.

Psychologically empowering actions can be taken by individual practitioners when a non-licensed reviewer de-

lays, alters, or refuses to authorize care. The practitioner should be attuned to the distinct possibility that this reviewer is practicing medicine without a license and report this person to state authorities for prosecution. While I urge the provider to exhaust the appeals process for any adverse reviewer decision, the first level reviewer must be held responsible.

Similarly, when licensed physicians in their roles as reviewers alter, delay, or refuse to authorize treatment, they should be reported for breaches of ethics ("do no harm") to their respective professional associations and licensing boards. Those who impact the morbidity and mortality of patients can and should be held responsible for their decisions. Any physician witnessing such destructive acts should be alert to this option.

Not only should this strategy increase your morale ("I can do something"), it can also alter the system. To be reported for an ethical violation is no laughing matter. Even if exonerated, the amount of paperwork, time, and emotional energy required of the accused in this process might deter the reviewer from continuing in this line of work, particularly if the number of complaints exceeds even one. As reviewers are called to account for their actions, the managed care industry might find it

harder to recruit them. Perhaps the system will not flow as smoothly because the law requires licensed reviewers at the appeal level.

The American Medical Association (AMA) can assist in this strategy by taking a deliberate, explicit stance on the legitimacy of this line of work. By way of precedent, the American Psychiatric Association's ethical principles³ explicitly rule out participation in a state sanctioned execution. The AMA could declare managed care review ethically unconscionable as well.

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Dr. Sank is a clinical psychologist in practice in Bethesda, Maryland.

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Gilbert B. Cushner, M.D., and his son **Fred Cushner, M.D.**, coauthored the chapter "Fluid Balance" in the text "Sports Medicine – Principles of Primary Care," recently published by Mosby. Dr. Gilbert Cushner is a clinical associate professor of medicine at George Washington University School of Medicine and is in private practice in Silver Spring. Dr. Fred Cushner was with the Greater Washington Orthopedic Group and is currently a director at the Insall Scott Kelly Institute for Orthopedics and Sports Medicine affiliated with Beth Israel Medical Center, North Division, in New York City.

Guohua Li, M.D., Dr.PH. is lead author in a study entitled "A Comparative Analysis of Alcohol in Fatal and Nonfatal Bicycling Injuries," published in the December issue of *Alcoholism: Clinical and Experimental Research* (1996;20:1553–1559). The results indicate that alcohol may have an even greater impact on cyclists than motorists. Dr. Li is an assistant professor of emergency medicine at The Johns Hopkins University School of Medicine. Other authors include **Susan P. Baker, M.P.H.** and **Sophia Sterling** also from Johns Hopkins; **John E. Smialek** from the office of the Chief Medical Examiner of Maryland; and **Patricia C. Dischinger** and **Carl A. Soderstrom, M.D.** from the University of Maryland School of Medicine.

A water-based pillow significantly improves quality of sleep and modestly reduces morning pain intensity and overall pain compared with other pillow types in people with benign neck pain, according to a study published in the February issue of *Archives of Physical Medicine and Rehabilitation*. Lead author **Robert A. Lavin, M.D.** stated that improved support may result from the pillow reducing head movement during sleep, conforming to changing positions of the head and neck, and absorbing and redistributing the weight of the head and neck. Dr. Lavin is assistant professor of physical medicine and rehabilitation at the Johns Hopkins Medical Institutions.

Michael Miller, M.D., presented the results of an 18-year study that shows triglyceride poses new risk for heart disease at the American Heart Association's 69th Scientific Sessions. At the same meeting, **Susan Bennett, M.D.**, reported on recent studies indicating that beta-blockers help improve heart function in people with heart failure. Dr. Miller and Dr. Bennett are from the University of Maryland Medical Center.

Herbert Chen, M.D., Martha A. Zeiger, M.D., Toby A. Gordon Sc.D., and Robert Udelsman, M.D. authored "Parathyroidectomy in Maryland: Effects of an endocrine center," published in the December issue of *Surgery* (1996;120:948–953). Authors concluded that patients with hyperparathyroidism are increasingly referred to an endocrine surgery center, resulting in high cure rates, low morbidity, no mortality, and a shorter hospital stay. Drs. Chen, Zeiger, Gordon, and Udelsman are from the division of endocrine and oncologic surgery, department of surgery, the Johns Hopkins Hospital.

Barry Meisenberg, M.D. is lead author in a study showing that high-dose chemotherapy, combined with a stem cell transplant, can be performed safely and effectively in an outpatient setting for many cancer patients. The study appears in the January issue of the *Journal of Clinical Oncology*. Dr. Meisenberg says outpatient therapy is possible because of new and better drugs to prevent infections and control the side effects of chemotherapy. Dr. Meisenberg is from the University of Maryland's Greenebaum Cancer Center.

Ethylin Jabs, M.D. is among the authors of a study identifying TWIST as the disease gene causing Saethre-Chotzen syndrome, one of the most common genetic conditions with craniosynostosis, the early closure of the cranial sutures. The findings appear in the January issue of *Nature Genetics*. Dr. Jabs is associate professor of pediatrics, medicine, and surgery, The Johns Hopkins Medical Institutions. Other study authors include members of Jab's lab at Hopkins, led by post-doctoral student **Timothy D. Howard, Ph.D.**

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Regional/local anesthesia: a safe and reasonable choice for patients undergoing carotid endarterectomy

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ABSTRACT

Purpose: The study compares the outcome of carotid endarterectomy in the community hospital setting using regional versus general anesthesia.

Methods: Two hundred thirty-six consecutive operations performed on 200 patients (99 operations using superficial and deep cervical block with local supplementation, and 137 procedures using general anesthesia) during a three-year period were analyzed retrospectively. Noncontinuous data were analyzed using Pearson chi-square, continuous data using Student's t-test.

Results: Demographic data and risk factors were similar for both groups. However, patients in the regional anesthesia group had a higher incidence of contralateral stroke and a lower incidence of peripheral vascular disease than patients in the general anesthesia group. Shunts were used less frequently for the regional anesthesia group. The neurologic complication rate was 2.2% for the general anesthesia group and 2.0% for the regional anesthesia group. The single death (fatal stroke) occurred in the general anesthesia group. Four of five major cardiopulmonary complications occurred in the general anesthesia group.

Conclusions: Carotid endarterectomy can be performed with an acceptable neurologic complication rate under either type of anesthesia. Use of regional anesthesia decreases intraoperative shunting and may decrease the rate of cardiopulmonary complications.

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Introduction

Following publication of the outcomes of several national and international trials,¹⁻⁴ carotid endarterectomy has experienced a renewed increase in popularity. Carotid endarterectomy at our institution increased 43% from 1990 to 1993.

The indications for carotid endarterectomy have become fairly uniform among vascular surgeons, but several issues regarding technique have not been resolved. Two of these issues are interrelated: whether the procedure should be performed on the awake patient and whether intraluminal shunting should be routinely utilized during carotid cross-clamping. Use of regional anesthesia obviates the need for routine shunting and eliminates the requirement for expensive intraoperative monitoring. However, controversy and emotion surround the performance of carotid endarterectomy on awake patients. Some surgeons are strongly opposed, while others embrace the technique. Reports either extol the virtues of carotid endarterectomy performed under regional anesthesia⁵⁻¹³ or vilify it.¹⁴⁻¹⁵ Most surgeons have been taught to perform this operation using general anesthesia, believing that there is increased cerebral protection, and that the operation performed with general anesthesia is easier on both surgeon and patient. However, advances in regional anesthesia and neuroleptic sedation have made this technique more comfortable for patients and surgeons alike.

We retrospectively analyzed a series of 236 consecutive carotid endarterectomies performed at our institution. This report compares the results of carotid endarterectomy performed using regional anesthesia to results of carotid endarterectomy performed using general anesthesia in a similar patient population at a single institution by a diverse group of 14 surgeons, half of whom performed at least one operation under both types of anesthesia during the 36-month period examined.

Material and methods

Data Collection. In preparation for this study, department of surgery personnel developed a code sheet for carotid endarterectomy. This form was designed for recording demographic data, risk factors, indications for operation, angiographic information, various intraoperative technique information, hospital stay information, and perioperative morbidity and mortality. After approval by the institutional review board, the instrument was used to audit the hospital records of 236 consecutive carotid endarterectomies performed on 200 patients at the Greater Baltimore Medical Center between July 1, 1990, and June 30, 1993. No patient undergoing carotid endarterectomy during this period was

omitted. Operations of carotid subclavian bypass and external carotid endarterectomy were excluded from data collection. In-hospital data were collected predominantly from a thorough review of hospital records, including angiographic reports and physicians' and nurses' notes. Confidentiality was ensured by assigning each patient and physician an identification number known only to the reviewer. Data were entered on a Wyse Decision 486/33T PC for statistical analysis. Analysis of data was accomplished using Pearson chi-square and Student's t-test for independent samples. A *P* value of .05 or less was considered to be significant.

Data on demographic information, risk factors, indications for surgery, intraoperative characteristics, and neurologic and non-neurologic complications were collected. Neurologic complication was defined as permanent (including fatal stroke) or transient (a deficit more than 24 hours but less than 30 days in duration).

Operative technique-general anesthesia group. Patients undergoing carotid endarterectomy were brought to the operating room. An arterial line was placed. Central venous pressure/pulmonary artery (CVP/PA) lines were not routinely used. However, in patients with severe cardiac dysfunction, these lines were placed prior to the administration of anesthesia. These were required in less than 2% of patients.

General anesthesia was induced in the following manner for a majority of patients: patients were oxygenated with 100% oxygen for 5 minutes. Midazolam 0.05mg/kg IV was given in conjunction with sublimaze 1-2 mcg/kg. Sodium pentothal 2.5% was next administered at 2 to 4 mg/kg. For those patients requiring muscle relaxation, succinylcholine was used (1.5 mg/kg) with non-depolarizing pretreatment (Curare 3 mgm IV). Following these medications, endotracheal intubation was accomplished under direct visualization. Patients were placed on a mixture of oxygen and nitrous oxide at 50:50 and isoflourane, unless contraindicated, at .8-2%. Respiration was controlled to maintain an end tidal CO₂ 30-40 torr (A torr is a unit of pressure equal to 1/760 of an atmosphere.). If hypotension occurred, patients were treated with dopamine 3 to 6 mcg/kg/min. Doses were titrated to maintain the patient's pressure. In patients with systolic blood pressures greater than 200 torr, the following protocol was undertaken: (1) increase of inhalation anesthesia; (2) intravenous narcotics; and (3) nitroglycerine 1 to 4 mcg/kg/min or labetalol was used commonly as a continuous infusion of 0.5-2 mgm/min. Intravenous heparin ranging from 3000 to 5000 units was administered prior to carotid cross-clamping. All patients were awakened in the operating room. The sterile field was not removed until the patient was satisfactorily awake and moving the contralateral side appropriately.

Operative technique-regional anesthesia group. Patients undergoing carotid endarterectomy were brought to the operating room. An arterial line was placed. A combination of superficial and deep cervical blocks was carried out by the anesthesiologist. Superficial cervical block was accomplished by injecting the posterior border of the sternocleidomastoid muscle under the external jugular vein in a fan-like manner with 15 ccs of a mixture of equal proportions of 1% xylocaine and .5% bupivacaine. Deep cervical block was accomplished by first drawing a line on the patient from the mastoid process to Chassaignac's tubercle. The transverse processes of C2-C4 lie along this line and are usually palpable. Using the same anesthetic agent, the needle is placed perpendicular to this line and slightly caudad, until each transverse process is contacted with the needle. The needle is aspirated for possible blood or cerebrospinal fluid. Three milliliters of local anesthesia is injected in each of these areas. During this procedure, patients are lightly sedated; however, they must be able to move and respond to questions at all times. Patients were administered 3000 to 5000 units of heparin prior to carotid artery cross-clamping. They were asked to move the hand opposite to the carotid artery being clamped every 1 to 2 minutes during the cross-clamping process. Patients remained awake throughout the entire surgical procedure, allowing continual assessment of their neurologic status.

Patients. Patients ranged in age from 40 to 89 years, with a mean of 69 years (SD = ± 9 years). There were 116 males and 84 females. Almost all patients were Caucasian (195). **Table 1** displays the demographic characteristics and risk factors of the patients by the type of anesthesia used.

Hypertension was present in 158 patients (79%), and coronary disease was present in 112 patients (56%); 40 patients (20%) had a history of myocardial infarction, 18 patients (9%) had a history of congestive heart failure, and 104 patients (52%) suffered from atherosclerotic heart disease. Sixty-one patients (31%) presented with history of stroke; 36 patients (18%) with a history of contralateral stroke, and 27 patients (14%) with a history of ipsilateral stroke (two patients had histories of both ipsilateral and contralateral strokes). Eighteen patients (9%) had a surgical history of contralateral carotid endarterectomy, and 6 patients (3%) had a previous ipsilateral carotid endarterectomy.

Table 1. Patient demographics and risk factors by type of anesthesia

	Total (n=200)	General (n=117)	Regional (n=83)
Average age (year)	69 (± 9)	69 (± 9)	67 (± 9)
Males/females (%)	58/42	58/42	58/42
Diabetes (%)	28	27	29
Hypertension (%)	79	74	86
Coronary disease (%)	56	55	58
MI (%)	20	21	19
CHF (%)	9	9	7
CAD (%)	52	50	54
Angina (%)	28	27	29
Stroke (%)	31	24	40*
Contralateral (%)	18	11	28†
Ipsilateral (%)	14	14	13
Peripheral vascular disease (%)	75	81	68‡
Hyperlipidemia (%)	37	43	32
Smoking (%)	77	76	77

*Pearson chi-square = 5.74; df = 1; p = .017

†Pearson chi-square = 9.06; df = 1; p = .003

‡Pearson chi-square = 4.95; df = 1; p = .026

Fifty-two patients (26%) were diabetic. There were several statistically significant differences noted for demographic and risk factors between the regional anesthesia and general anesthesia groups. A higher percentage of patients in the regional anesthesia group had a history of contralateral stroke prior to carotid endarterectomy, while a higher percentage of patients in the general anesthesia group had a history of peripheral vascular disease.

The percentage of stenosis was measured angiographically, by comparing the smallest transverse diameter at the point of maximal stenosis with the diameter of the postbulbular internal carotid artery, at the point its diameter was uniform. Of all carotid endarterectomies, 90% had stenoses $\geq 70\%$, and the mean percentage of carotid stenosis for both groups was 85%; 90% of the general anesthesia group and 92% of the regional anesthesia group had lesions $\geq 70\%$. However, 37% of the general anesthesia group as opposed to 23% of regional anesthesia group were asymptomatic (p=.030). Of all lesions, 139 (59%) were found to be ulcerated by examination of the pathology specimen.

Table 2 summarizes the indications for carotid endarterectomy for all operations and contrasts indications for the general anesthesia and regional anesthesia groups. Stroke was an indication for carotid endarterectomy for 21%

Table 2. Indications for carotid endarterectomy for all operations by type of anesthesia*

	Total (n=236)	General (n=137)	Regional (n=99)
TIA (%)	49	49	50
Crescendo TIA (%)	2	2	3
RIND (%)	3	4	3
Stroke (%)	14	9	21 [†]
Asymptomatic bruit (%)	31	37	23 [‡]

*Percentages may not total 100 due to rounding

[†]Pearson chi-square = 6.42; df = 1; p = .011

[‡]Pearson chi-square = 4.73; df = 1; p = .030

of the regional anesthesia group, but for only 9% of the general anesthesia group (p=.011).

Results

There was one death in this series for an operative mortality rate of 0.4% (1/236). The single mortality was due to massive stroke and occurred in the general anesthesia group. Perioperative neurological morbidity included transient deficit in three patients (1.3%) and permanent deficit in one patient (0.4%), for a stroke/mortality rate of 2.1% (5/236). **Table 3** compares the outcomes for both groups. We were able to follow 177 patients for more than 30 days postoperatively and found there were no new neurologic events or deaths during that time. The remaining 23 patients were lost to follow-up.

There were five major cardiopulmonary complications, four of which occurred in the general anesthesia group. In the general anesthesia group, one patient had a myocardial infarction, one patient suffered pulmonary edema, one patient experienced ventricular ectopy, and one patient experienced postoperative angina. The patient in the regional anesthesia group suffered congestive heart failure. None of these complications resulted in a fatality.

Intraoperative shunts were used in 47 of the operations performed using general anesthesia (34%), but in only 11 of the operations (11%) performed using regional anesthesia (p ≤ .001). The mean occlusion time for the general anesthesia group was 19 minutes, compared to 38 minutes for the regional anesthesia group (p < .001), a difference we attribute to performing the operation on an awake patient, allowing the surgeon to proceed at a more leisurely pace, since the

awake patient's neurological status is never in question. Mean length of stay in intensive care was not significantly different for the two groups (general anesthesia=1.3 days; regional anesthesia=1.5 days; p=.14). Mean postoperative hospital length of stay was also approximately equal for the two groups (general anesthesia = 3.9 days; regional anesthesia = 4.4 days; p = .47).

A clear relationship emerged between postoperative hypertension and poorly controlled pre-operative hypertension. Poorly controlled pre-operative hypertension was defined as a systolic blood pressure equal to or exceeding 160 mm Hg during the immediate pre-operative period. A total of 53 carotid endarterectomies (23%) were performed on patients who fit this classification, 23 carotid endarterectomies (17%) in the general anesthesia group and 30 carotid endarterectomies (30%) in the regional anesthesia group (p=.014). Postoperative hypertension, defined as systolic blood pressure which equaled or exceeded 200 mm Hg and occurred within the first 48-hour period following operation, was experienced by 65 patients undergoing carotid endarterectomies (28%). More than half of the procedures performed on patients with poorly controlled pre-operative hypertension (51%) resulted in postoperative hypertension, compared to only 21% of carotid endarterectomies performed on normotensive patients (p ≤ .001). We failed to find a relationship between type of anesthesia and postoperative hypertension, however. Postoperative hypertension resulted in 26% of general anesthesia carotid endarterecto-

Table 3. Outcomes of carotid endarterectomy for all operations by type of anesthesia

	Total (n=236)	General (n=137)	Regional (n=99)
♦ Neurologic complications			
Any stroke/mortality (%)	2.1	2.2	2.0
Fatal stroke (%)	0.4	0.7	0.0
Permanent deficits (%)	0.4	0.7	0.0
Transient deficits (%)	1.3	0.7	2.0
♦ Cardiopulmonary complications			
All cardiopulmonary (%)	2.1	2.9	1.0
Angina (%)	0.4	0.7	0.0
Congestive heart failure (%)	0.4	0.0	1.0
Myocardial infarction (%)	0.4	0.7	0.0
Pulmonary edema (%)	0.4	0.7	0.0
Ventricular ectopy (%)	0.4	0.7	0.0

mies and 29% of regional anesthesia carotid endarterectomies ($p=.609$). In the general anesthesia group, 21% of carotid endarterectomies performed on normotensive patients and 52% of carotid endarterectomies performed on poorly controlled hypertensive patients resulted in postoperative hypertension; in the regional anesthesia group, 20% of carotid endarterectomies performed on normotensive patients and 50% of carotid endarterectomies performed on poorly controlled hypertensive patients resulted in postoperative hypertension.

Discussion

We failed to find a relationship between type of anesthesia used and frequency of postoperative neurologic complications. This is most likely due to the small number of patients in this series who experienced such complications, rather than an indication that no relationship between type of anesthesia and postoperative neurological complication rate exists.

Cerebral protection is a major consideration when performing carotid endarterectomy. Mechanical protection, as opposed to pharmacologic protection, is primarily furnished through the use of a shunt during the surgical procedure. Although a shunt maintains blood flow through the carotid artery, its use is not free of potential injury. The use of arterial shunts has been correlated with intimal dissection, air embolism, thromboembolism, thrombus formation, and obstruction of the surgical field.²² Our findings support the conclusion that performing carotid endarterectomy under regional anesthesia assists in identifying those patients who require the use of shunts, thereby decreasing the number of patients subjected to the potential sequelae associated with their use.

Monitoring the adequacy of cerebral perfusion in the patient under general anesthesia is a dilemma that lacks reliable resolution. Stump pressure, electroencephalogram, somatosensory evoked potential, measurement of cerebral blood flow by xenon washout, and measurement of jugular venous oxygen saturations are various methods which have been evaluated for assessing satisfactory cerebral blood flow. Xenon washout, jugular venous bulb measurements, and stump pressure measurements have been demonstrated to be unreliable identifiers of patients at risk for developing cerebral ischemia.²³ Although the unprocessed electroencephalogram has been reported as a reliable predictor of ischemia, in patients who have previously sustained central nervous system insult (stroke or reversible ischemic neurological deficit) it has proven less reliable.²² Both the unproc-

essed and processed electroencephalogram reflect only surface area activity of the brain and can miss ischemic processes affecting deep structures. Additionally, both are affected by many other factors: serum glucose, serum electrolytes, body temperature, anesthetic and premedicant drugs, arterial oxygenation and carbon dioxide.²² Compared to the limitations of expensive intraoperative monitoring equipment used to evaluate the adequacy of cerebral blood flow in patients receiving general anesthesia for carotid endarterectomy, assessing changes in motor ability, mentation or level of consciousness in patients undergoing carotid endarterectomy using local/regional anesthesia is a simple and trustworthy method.

Because of its apparent protective effects on the central nervous system, isoflurane has been recommended as the anesthetic of choice for patients undergoing carotid endarterectomy. However, many patients have coexistent coronary artery disease and carotid artery disease, and patients at risk for coronary artery disease cannot always be readily identified.²⁴ Use of isoflurane in patients with ischemic heart disease has been demonstrated to produce a "steal," resulting in increased lactate production. Isoflurane may promote focal areas of ischemia in this population.²⁵⁻²⁷ Use of regional anesthesia not only permits accurate assessment of cerebral perfusion, but allows detection of myocardial ischemia symptoms as well.

There is some question in the literature as to whether regional anesthesia is correlated with a lower incidence of peri-operative myocardial infarction.^{11,28-30} While our data are not directly relevant to this consideration, our results tend to support the position indirectly. Although failing to achieve statistical significance, four of the five major cardiopulmonary complications occurring in this series of patients occurred in the general anesthesia group.

Postoperative neurologic deficits have been reported to be more common in those patients who experience postoperative hypertension,^{13,16,17} while other investigators have found no difference in the incidence of neurologic sequelae between normotensive and hypertensive patients.¹⁸⁻²¹ Corson and others⁶ reported that patients who received regional anesthesia for carotid endarterectomy required a significantly shorter period of treatment with intravenous vasodilator drugs for hypertension than those who received general anesthesia for their operation. Unlike Corson, we found no relationship between type of anesthesia used and postoperative hypertension. In this instance, although the measure of hypertension used was sufficient to establish a relationship between poorly controlled pre-operative hypertension and

the occurrence of postoperative hypertension, the measure of hypertension used was less than optimal. It is the observation of the authors that there has been a significant decrease in the incidence of immediate postoperative hypertension reflected by a decrease in necessary postoperative antihypertensive medication in the patients operated upon under cervical block. This comparison will be the subject of a prospective study, and further research in this direction will proceed using more refined measurement criteria.

Conclusions

Regional anesthesia for carotid endarterectomy offers many actual and potential benefits when contrasted with general anesthesia. It permits more direct and less expensive assessment of the adequacy of cerebral blood flow, and patients requiring the use of temporary carotid shunts can be readily identified. Intraoperative and postoperative neurologic evaluations can be performed more quickly and reliably.

The use of regional anesthesia is safe and well-tolerated by the patient. These data demonstrate that carotid endarterectomy can be performed with an acceptable neurologic complication rate under either regional or general anesthesia. Use of regional anesthesia decreases the need for intraoperative shunting, and may decrease the rate of cardiopulmonary complications associated with carotid endarterectomy, as well as the volume and duration of use of intravenous vasodilators in the treatment of postoperative hypertension.

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CASE RECORDS

of the *University of Maryland and Baltimore VA Medical Centers.*

A 73-year-old woman with hemoptysis and shortness of breath

Carl Shanholtz, M.D., discussant

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PRESENTATION OF CASE

A 73-year-old woman with a four-month history of intermittent hemoptysis presented to a hospital. On presentation she had a cough, and a chest x-ray showed bilateral lower lobe infiltrates (Figure 1). She was treated with a first generation cephalosporin and subsequently with a quinolone antibiotic resulting in a decrease in the amount of hemoptysis. At 48 hours there was no response to a 5 tuberculin unit purified protein derivative, but an "anergy panel" was not placed.

Her medical history was remarkable for ovarian cancer treated, 13 years prior to presentation, with a combination of surgical resection and "a pill." She had never smoked. Her father died of tuberculosis when she was age 6, but no one else in the family had ever had tuberculosis. She had been treated with a diuretic for hypertension.

Because of continued hemoptysis despite antibiotic therapy, fiberoptic bronchoscopy was performed. It showed acute and chronic inflammatory changes without evidence of granuloma or infection. The hemoptysis continued and bronchoscopy was repeated. In the bron-

chus intermedius a "heaped up ridge" of tissue with slight inflammation, denuded mucosa, and submucosal hemorrhage was noted, and a biopsy was performed. The pathologic specimen showed inflammation dominated by neutrophils, mild focal thickening with fibrosis, chronic inflammatory changes and reactive pneumocytes. Fluid from bronchoalveolar lavage was negative for acid-fast bacilli by smear, but *Mycobacterium avium* complex was grown in culture. She was treated for one month with clarithromycin and ethambutol. Despite treatment she became increasingly dyspneic, especially with exertion. She continued to cough, with intermittent hemoptysis and production of white frothy sputum. Two days prior to admission she became markedly more short of breath and developed chest pain. Her chest x-ray at the referring hospital showed a diffuse interstitial pattern, involving the right lung more than the left, and obscuring both diaphragms. A ventilation-perfusion scan of the lungs was indeterminate in probability for thromboembolic disease, but there was no evidence of deep venous thrombosis of the lower extremities by ultrasound examination. A pulmonary angiogram showed no evidence of pulmonary embolism. There was no electrocardiogram (EKG) or enzymatic evidence of myocardial



Figure 1A.



Figure 1B.

CASE RECORDS

infarction. She was transferred to the University of Maryland Medical System (UMMS) for further evaluation.

On presentation to UMMS her blood pressure was 142/78 mm Hg, and her pulse was 98 and regular. The patient was breathing 18 times per minute, and her saturation of oxygen on four liters of inspired oxygen by nasal canula was 93%. Her temperature was 99°F. No adenopathy was present. Examination of the lungs showed diffuse coarse rhonchi, worse at the bases, without wheezing or egophony. The patient exhibited good air movement. Her cardiovascular exam was unremarkable. She was mildly obese, but her abdominal exam was otherwise normal. There was no edema or cyanosis of the extremities. Admission laboratory values are shown in **Table 1**. A computed tomogram of the chest showed diffuse airspace disease, right worse than left (**Figure 2**). A diagnostic procedure was performed.

DIFFERENTIAL DIAGNOSIS

There are over one hundred different causes of hemoptysis, and I've been challenged to use the information presented in this case to narrow down the list of suspected etiologies.

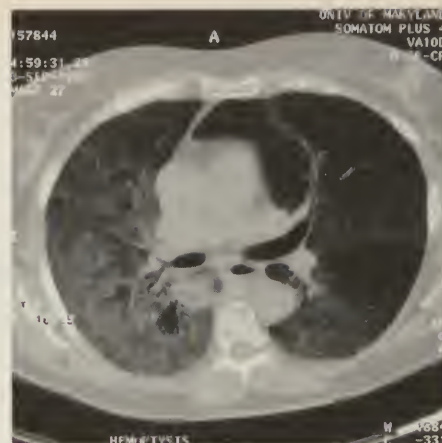


Figure 2.

First, let's review the salient features of this case (**Table 2**). The chief presenting symptom was hemoptysis, or expectoration of blood. Quantities of blood can range from blood-streaked sputum to massive hemoptysis, defined as 600 ml of expectorated blood in 24 hours. To understand where bleeding can occur, it is helpful to review the dual circulation of the lung. The pulmonary circulation originates from the right side of the heart, is a low pressure circuit, serves its primary function of gas exchange, and is returned to the left side of the heart. The bronchial circulation originates from the systemic circulation (either thoracic aorta or intercostal arteries), supplies nutrition to the bronchovascular tree, anastomoses at the capillary level with the pulmonary circulation, and accounts for a fixed amount of right-to-left intrapulmonary shunt.¹

Although the bronchial arteries are small vessels, they are under higher pressures than the pulmonary arteries, and bronchial arterial bleeding can be massive.

The differential diagnosis of hemoptysis is lengthy, yet some disease processes can be categorized for conceptual purposes (**Table 3**). I'd like to immediately eliminate the diagnoses in the right column. We have reason to suspect that this patient had lung disease and no reason to believe that the source of bleeding was outside of the lungs, so we can dispense with pseudohemoptysis. A myocardial infarction was ruled out, and her physical examination and radiographs

Table 1: Laboratory values

• WBC	11,600/cc ³ - 66% segmented cells, 15% lymphocytes, 6% mononuclear cells, 13% bands		
• Hematocrit	41.9%	• Platelets	192,000/cc ³
• Serum Sodium	138 mg/dl	• Serum Potassium	3.4 mg/dl
• Serum Bicarbonate	29 mg/dl	• Serum Chloride	99 mg/dl
• Blood Urea Nitrogen	8 mg/dl	• Serum Creatinine	0.8 mg/dl
• Serum Glucose	141 mg/dl		
• Prothrombin time	10.1	• Partial thromboplastin time ..	24
• AST	19 units/L	• Alkaline phosphatase	79 units/L
• ALT	22 units/L	• LDH	208 units/L

Pulmonary Function Tests:

	3 months prior to admission	2 weeks prior to admission
• FEV ₁	140% predicted	100% predicted
• FEV ₁ /FVC	83%	85%
• DLCO	61% predicted	55% predicted

were not consistent with congestive heart failure; so, I don't suspect there was a cardiogenic cause for hemoptysis. Her laboratory studies revealed no evidence of a coagulopathy. Negative pulmonary angiography and Doppler flow studies of the lower extremities following indeterminant ventilation-perfusion radionuclide scanning effectively rule out thromboembolic disease. It is unlikely that a congenital structural abnormality, such as an arteriovenous malformation or Rendu-Osler-Webersyndrome, would present in the seventh decade of life. Idiopathic pulmonary fibrosis is a rare cause of hemoptysis and this patient had restrictive physiology. However, she did not have diffuse crackles on examination, and the radiographs showed dense consolidation rather than interstitial infiltrates.

I will discuss the disease entities in the left column of **Table 3** in more detail. Malignancy is the most common cause of hemoptysis today, with bronchogenic carcinoma accounting for nearly 30% of the cases.² Bronchitis is the second most common cause accounting for 23% of the cases, followed closely by idiopathic hemoptysis. The other diagnostic categories are less frequent causes of hemoptysis, but should be included in the differential diagnosis of this case.

Diseases that cause pulmonary vasculitis are listed in **Table 4**. Wegener's granulomatosis is a systemic disease characterized by a triad of necrotizing granulomas of the respiratory tract, necrotizing vasculitis of the arteries and veins of the lung, and glomerulonephritis. The disease usually occurs in the fifth decade of life, predominantly affects men, and often affects organs other than the lungs. Antineutrophil cytoplasmic antibodies (ANCA) present in the blood have been associated with vasculitides. The cytoplasmic ANCA (c-ANCA) is most specific for Wegener's granulomatosis, whereas the perinuclear ANCA (p-ANCA) has been found in other vasculitides.³ There was no evidence of renal dysfunction or other systemic disease, and thus it is unlikely that this patient had Wegener's granulomatosis. Churg-Strauss syndrome is an allergic granulomatosis and vasculitis associated with eosinophilic pulmonary infiltrates and peripheral eosinophilia.

Table 2: Salient features

- Hemoptysis
- Progressive dyspnea
- Remote history of ovarian cancer
- Family history of lung cancer
- PPD negative—no anergy panel. Remote TB exposure
- Bronchoscopy x 2: inflammation, *M. avium* complex grown
- Diffuse interstitial disease on plain chest radiograph. Dense consolidation by CT
- V/Q scan indeterminant; LE Dopplers and pulmonary angiography negative

Table 3: Causes of hemoptysis

- | | |
|--------------------------|---------------------------------------|
| • Malignancy | • Idiopathic pulmonary fibrosis |
| • Bronchitis | • Congenital structural abnormalities |
| • Idiopathic | • Pulmonary thromboembolic disease |
| • Infection | • Coagulopathy |
| • Sarcoidosis | • Cardiogenic |
| • Goodpasture's syndrome | • Pseudo-hemoptysis |
| • Vasculitis | |

The major presenting pulmonary symptom is asthma, and it is a rare cause of hemoptysis. Lymphomatoid granulomatosis is a rare lymphoproliferative disorder associated with nodular infiltrates on chest roentgenograms. Hemoptysis is more common in patients with cavitary lesions, and we can exclude this diagnosis as an unusual presentation of an unusual disease. Although Behçet's disease may have pulmonary involvement associated with massive hemoptysis, the disease is characterized by aphthous stomatitis and possibly genital ulcerations, uveitis, cutaneous nodules, or synovitis, none of which were mentioned in the presentation. Henoch-Schönlein purpura is a syndrome with the triad of palpable purpura, arthritis, and colicky abdominal pain. Pulmonary involvement is rare. Leukocytoclastic vasculitis, mixed cryoglobulinemia, and connective tissue disease also have systemic manifestations which are absent in this patient.

Goodpasture's syndrome is a pulmonary alveolar hemorrhage syndrome which should be considered in

Table 4: Pulmonary vasculitides

- Wegener's granulomatosis
- Churg-Strauss syndrome
- Lymphomatoid granulomatosis
- Behçet's disease
- Henoch-Schönlein purpura
- Leukocytoclastic vasculitis
- Mixed cryoglobulinemia
- Connective tissue disease

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the differential diagnosis of a patient presenting with hemoptysis. The mechanism of disease is the destruction of type IV collagen in the capillary wall caused by antiglomerular basement membrane (anti-GBM) autoantibodies. As the name implies, the primary target for the antibodies is the kidney, and the disease is associated with a rapidly progressive glomerulonephritis. The typical chest roentgenographic findings of Goodpasture's syndrome are bilat-

frequency over the years² presumably due to the development of antibiotics to treat recurrent infections. This patient did not demonstrate radiographic evidence of inappropriately widened airways seen in bronchiectasis. Although her history reveals transbronchial biopsy evidence of inflammation, and her admission laboratory data demonstrate a mildly elevated white blood cell count with a left shift, she was afebrile on admission, and we are given no culture evidence for a bacterial pneumonia. Fungal disease is unlikely in the absence of immunocompromise and exposure history. However, some issues related to mycobacterial disease are worth mentioning in light of this patient's history. She had a remote exposure to tuberculosis in childhood, and a PPD was done which was negative, although an anergy panel was not placed. Up to 3% of patients with proven tuberculosis may have a nonreactive skin test to tuberculin, and an anergy panel may not accurately define these cases, since about half may be associated with skin tests that react to nonmycobacterial antigens.⁶ Cutaneous reactivity may be altered by old age and other conditions, and negative tuberculin reactions can be seen in primary or overwhelming tuberculosis infections. This patient had hemoptysis and radiologic consolidation which refutes a diagnosis of primary tuberculosis, and it is unlikely that bronchoscopy would not yield a diagnosis if this were advanced disease. Although I am always careful about turning my back on tuberculosis, I believe this diagnosis is unlikely.

According to the history, *Mycobacterium avium* complex was grown in culture from a bronchoalveolar lavage. Although pulmonary disease due to *Mycobacterium avium* complex is commonly associated with acquired immunodeficiency syndrome and elderly men with underlying chronic lung disease, there have been reports of *Mycobacterium avium* complex producing disease in patients without predisposing risk factors.^{7,8} Twenty-one such patients were reported in the *New England Journal of Medicine* by David Prince, a former chief resident from our program, and others.⁷ They described the disease as indolent with cough being present a mean of 25 weeks prior to diagnosis, and radiographic disease progression occurring over at least two to three years.

Table 5: Staging of sarcoidosis

Stage	Radiographic abnormality	% presenting
0	None	Up to 8
I	Hilar, mediastinal, paratracheal adenopathy	40-50
II	Adenopathy and pulmonary abnormalities	15-30
III	Pulmonary abnormalities without adenopathy	10-15

eral, diffuse, perihilar infiltrates with sparing of the costophrenic angles. When the lungs are affected in anti-GBM disease, it is usually in young men in their twenties. Elderly women more commonly get renal disease alone. The absence of renal disease in this patient, as well as her gender, age, and the radiographic appearance of the infiltrates, make Goodpasture's syndrome unlikely.

Sarcoidosis can be included in the differential diagnosis of many pulmonary disease presentations because of the marked heterogeneity of symptoms and findings. The staging of sarcoidosis is shown in Table 5. This patient's radiographic presentation with peripheral parenchymal infiltrates and absent hilar or mediastinal adenopathy would be considered stage III disease. Although a few patients will present with advanced disease, only 1% to 3% of all sarcoid patients initially present with hemoptysis. Radiographic findings in sarcoidosis are more commonly symmetrical and diffuse with an upper lobe or middle lung field predominance, and end stage disease is associated with fibrosis.^{4,5} Bronchoscopy and transbronchial biopsy are diagnostic in 80% to 90% of patients with stage III disease, which also refutes a diagnosis of sarcoidosis.⁴

Infectious causes of hemoptysis can be grouped into several categories (Table 6). Bronchiectasis, classically one of the most common causes of hemoptysis, has declined in

In addition, hemoptysis was uncommon, and the radiographic pattern was most commonly discrete nodules, with consolidation in only 10% of patients. Therefore, it is unlikely that the cause of hemoptysis and progressive pulmonary disease in our patient was *Mycobacterium avium* complex.

Cryptogenic, or idiopathic, hemoptysis accounts for over 20% of the cases of hemoptysis.² A description of 67 patients with cryptogenic hemoptysis co-authored by two of our faculty members found the prognosis of these patients to be generally good, with 85% demonstrating no evidence of tuberculosis or bronchogenic carcinoma over an average three-year period of follow-up.⁹ However, we cannot assume our patient had cryptogenic hemoptysis in the presence of radiographic abnormalities and progressive physiologic deterioration. Similarly, bronchitis is among the most common causes of hemoptysis.² Although bronchoscopy in our patient revealed inflammatory changes, it is difficult to ascribe hemoptysis to bronchitis in this patient who never smoked and who had progressive infiltrates.

Many malignancies, both hematologic and solid tumor, are associated with intrathoracic disease. I'll discuss amyloidosis with the hematologic malignancies because it tends to be a plasma cell disorder. The lungs are commonly affected in primary amyloidosis, whereas pulmonary disease is rare in secondary amyloidosis.¹⁰ The patterns of pulmonary involvement may be tracheobronchial or parenchymal, and nodular or diffuse.^{10,11} Pulmonary amyloidosis tends to be indolent,^{10,11} and an aggressive, diffuse alveolar-septal pattern that would fit our patient's presentation would also be associated with systemic disease,^{11,12} making this diagnosis unlikely.

Although thoracic presentation in lymphoma is common, the lung parenchyma is involved usually by direct extension from the hilum.¹³ Primary parenchymal Hodgkin's disease is rare, and our patient clearly had no hilar or mediastinal adenopathy. Our patient had diffuse airspace disease rather than the nodular, discrete, parenchymal lesions of lymphoma. Furthermore, bronchial mucosa-associated lymphoid tissue, which can account for extranodal disease, is indolent and remains localized.

Metastatic cancer frequently involves the lungs. The malignancies most likely to metastasize to the lungs include carcinomas of the colon, kidney, breast, testis, uterus, head and neck, and ovaries, as well as sarcomas and melanomas.¹⁴ Parenchymal patterns of metastatic cancer can be nodular or lymphangitic. The latter is characterized by a reticulonodular pattern and usually associated with breast, pancreatic, and gastric cancers.¹⁵ Our patient had a history of ovarian cancer. However, ovarian cancers are usually advanced when pulmonary metastases develop, and the patient's physical examination revealed no abdominal distention or evidence of ascites. She was treated for her ovarian cancer with surgical resection and an oral medication. The only oral adjuvant chemotherapy trials for ovarian cancer that were active around the time of her treatment were with oral melphalan in stage I and stage II disease, in which disease-free survival is high.¹⁶ Recurrence of ovarian cancer in these trials tended to occur within the first two years, and all occurred with seven years. Furthermore, the development of a new lung opacity in a patient with known extrathoracic malignancy is more likely to be a bronchogenic carcinoma.¹⁵ Therefore, metastatic cancer is unlikely to be the cause of her pulmonary disease.

Bronchogenic carcinomas arise in the airways. The four major histologies, squamous cell, adenocarcinoma, large cell anaplastic, and small cell carcinoma, tend to present as nodular or mass lesions. Squamous cell and small cell carcinomas frequently present as central masses, whereas adenocarcinomas and large cell anaplastic carcinomas are more frequently peripheral masses.¹⁷ Hilar or mediastinal adenopathy is commonly seen with these histologies. However, the salient features of our patient's computed tomographic studies of the chest were: dense airspace disease, air bronchograms, and the absence of hilar or mediastinal adenopathy. Furthermore, her spirometry

Table 6: Infectious causes of hemoptysis

- Bronchiectasis
- Pneumonia
- Lung abscess
- Fungus
- Aspergillus
- Coccidioidomycosis
- Cryptococcus
- Histoplasmosis
- Blastomycosis
- Mycobacteria
- Tuberculosis
- *Mycobacterium avium* complex

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was consistent with a progressive restrictive pattern rather than obstructive airways disease.

Bronchioloalveolar carcinoma, an uncommon histology whose cell of origin is the type II pneumocyte, closely fits this patient's presentation. Bronchioloalveolar carcinoma represents 1.5% to 6.5% of primary lung cancers.^{17,18} The average age of most patients is 54 years with a range of 40 to 70 years of age. Women are disproportionately affected compared to the other lung cancers, comprising 30% to 50% of the patients.¹⁸ Smoking is less commonly associated with bronchioloalveolar carcinoma than the other bronchogenic carcinomas, with 25% to 50% of patients being non-smokers or mild smokers.^{17,18} There is an association with chronic inflammation.¹⁸ The most common symptom is cough which is present in 70% of patients.¹⁸ Chest pain, sputum production, and dyspnea are also common. Hemoptysis occurs in 8% of patients.¹⁸ The radiographic pattern can be either a solitary, well-circumscribed pulmonary nodule, or diffuse airspace disease.^{17,19} The prognosis of solitary bronchioloalveolar carcinoma is good with a 30% to 70% five-year survival rate after resection. However, the prognosis of diffuse disease, which unfortunately our patient was more likely to have, is dismal with almost no patients surviving 3 years even if resected.¹⁸

It is not unusual that fiberoptic bronchoscopy with transbronchial biopsy was unable to yield a diagnosis since bronchioloalveolar carcinoma is a disease of airspaces, and there are no endobronchial lesions.¹⁸ There are two ways in which I would attempt to make a diagnosis. The first is by radiographically guided percutaneous transthoracic needle biopsy. This technique is

useful for peripheral lesions, or where the only alternative is a thoracotomy, and the patient is unlikely to be an operable candidate even if the lesion is proven to be malignant.²⁰ Alternatively, Dr. Krasna has published our institution's experience with thorascopic lung biopsy,²¹ which provides the advantages of direct visualization of the diseased area of the lung and the ability to obtain adequate tissue for diagnosis. This would have been a reasonable diagnostic procedure to perform on our patient who had already had nondiagnostic transbronchial forceps and Wang needle biopsies, and it carries less risk than a standard thoracotomy.

CLINICAL DIAGNOSIS: Bronchioloalveolar carcinoma. The diagnostic procedure of choice would either have been a percutaneous transthoracic needle biopsy or a thorascopic lung biopsy.

PATHOLOGIC DIAGNOSIS (Dr. Wolfgang Mergner, professor of pathology, UMMS): The sections are from a thorascopic lung biopsy and the findings are consistent with a diagnosis of bronchioloalveolar carcinoma. There is an inflammatory infiltrate in the background, and one notes the growth of well-differentiated cuboidal and columnar cells along alveolar walls as a single layer at low magnification (**Figure 3**). The line of tumor cells are attached to the alveolar wall. Some regions show papillary growth which protrudes into the alveolar space (**Figure 4**). The cells are more cuboidal in the non-mucinous bronchioloalveolar carcinoma and contain large nuclei. Most non-mucinous bronchioloalveolar carcinomas create some thickening of the interstitium of the alveolar wall, and this thickening may expand to more prominent interstitial fibrosis.

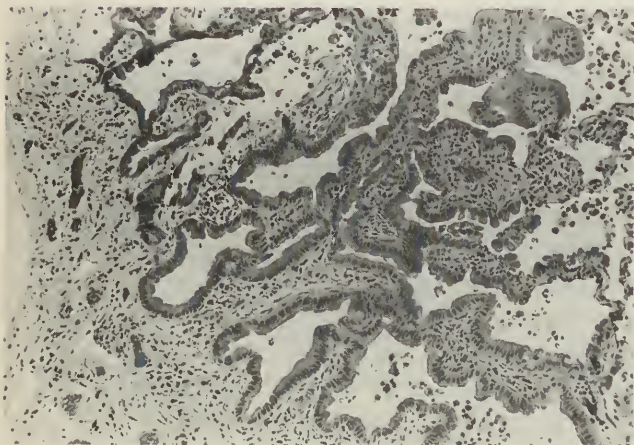


Figure 3.

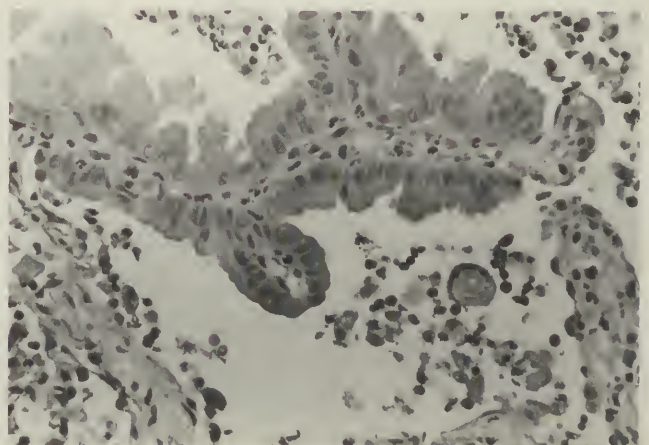


Figure 4.

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Med Chi's presidents: a series of historical vignettes

An institution's continuing growth depends largely upon the caliber of people who serve as its leaders. Many of Med Chi's presidents were among the best practitioners of their times. This series of historical vignettes will provide insight into the capabilities of these leaders.

THE BEGINNING

In 1799, in the spirit of cooperation, a group of Maryland doctors gathered in Annapolis and formed a state society – The Medical and Chirurgical Faculty of the State of Maryland (Med Chi). Little did the founding members realize that the organization they created would reach the age of 200. Troubles in the adolescent years were many, but the tact and perseverance of the men who served as leaders cured the serious maladies with which Med Chi was at times affected.

At the first meeting of 101 incorporators, many of whom were dominant medical individuals in their respective communities, Upton Scott, M.D., of Annapolis was apparently thought to be the most suited, by age, character, and experience, to lead the new organization. When his name was proposed, he declined the honor by reason of age. (He was

73.) He was, nevertheless, chosen unanimously and he accepted.

Scott, born in Ireland, graduated with a Doctor of Medicine from the University of Glasgow in 1753. He came to Maryland in 1753 and held a number of offices in the colonial government. Favored by the patronage of the Governor, he became court physician of Annapolis and enjoyed a large practice.

During the revolution, Scott sided with Great Britain and became a voluntary exile in Belfast and London. He subsequently returned to Maryland but did not resume the practice of medicine. Recalled as a man of high honor and integrity, he was deeply interested in medical progress.

Joseph M. Miller, M.D.

Dr. Miller is a retired surgeon in Timonium, Maryland.

AIM HIGH

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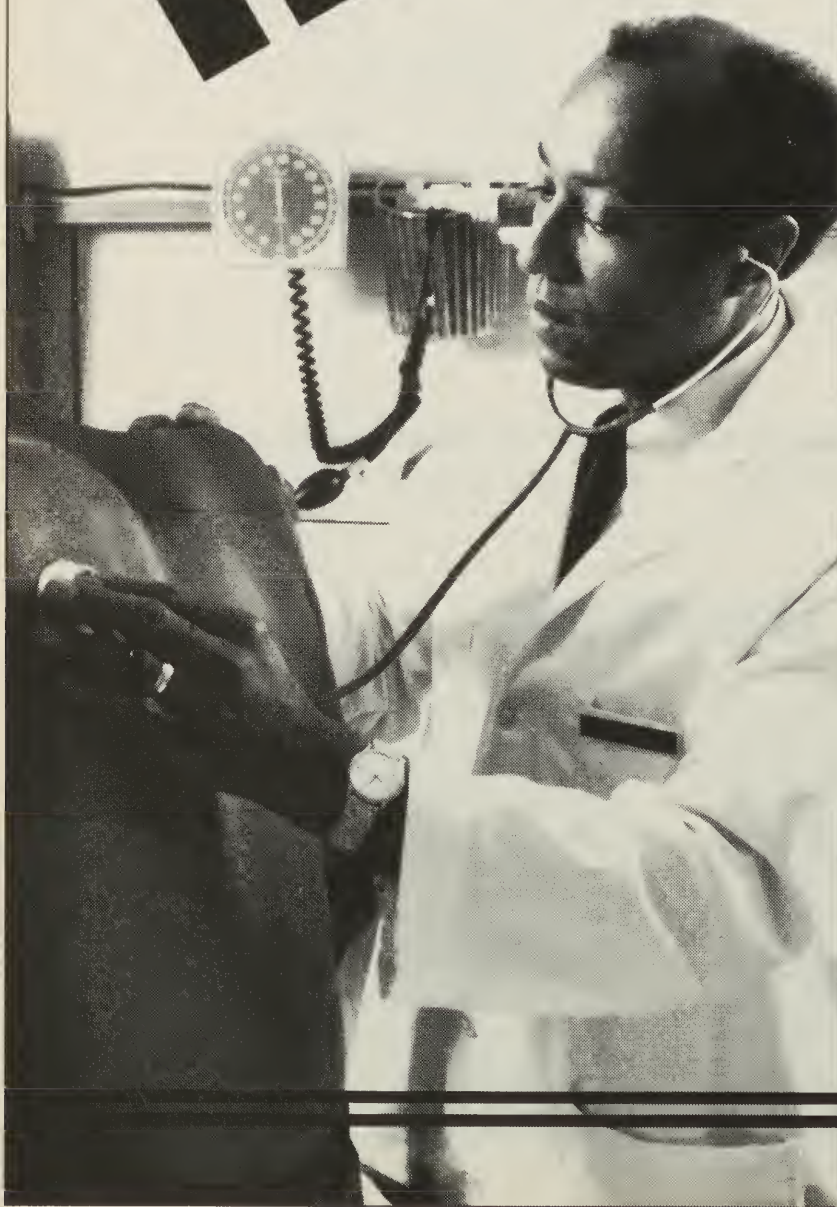
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Imaging Case of the Month

Extensive pneumatosis intestinalis and portal venous air developing after bowel infarction

Clinical history

A 72-year-old man with history of esophageal carcinoma underwent esophagectomy and gastric pull-up surgery. He was recovering uneventfully until he developed a sudden onset of severe abdominal pain and fever approximately 48 hours after surgery.

Imaging findings

An upright abdominal film demonstrates midline abdominal and right lower thoracic skin staples, multiple air/fluid levels throughout a minimally dilated small bowel, residual contrast in the stomach and proximal small bowel, and linear air radiodensities overlying the right upper quadrant (Figure 1A). A computed tomography (CT) scan shows extensive pneumatosis intestinalis throughout the visualized small bowel (Figure 1B) and portal venous air in the liver and portal vein (Figure 1C). Venous air was also visible within the superior mesenteric vein on CT cuts immediately inferior to the image depicted by Figure 1C.



Figure 1B. Abdominal CT demonstrates extensive pneumatosis intestinalis throughout the visualized small bowel (arrowheads).

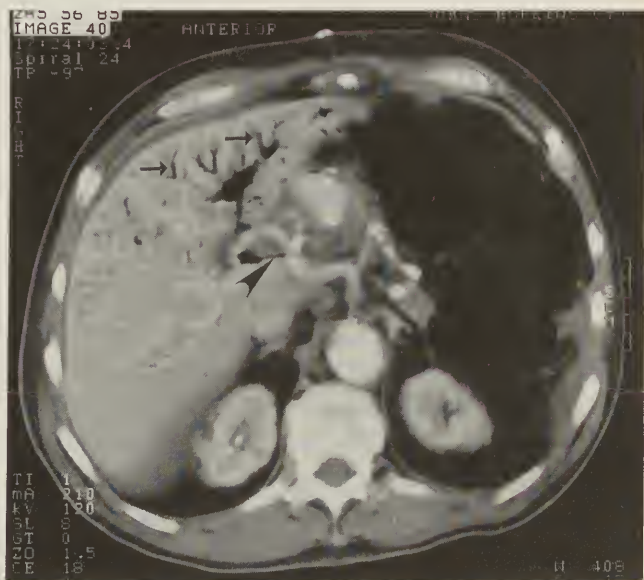


Figure 1C. Abdominal CT demonstrating extensive portal venous air in an antidependent location within the liver (arrows) and also tracking along the ventral portion of the portal vein into the portal venous confluence (arrowhead). Cuts immediately inferior to this position demonstrated air present within the superior mesenteric vein.



Figure 1A. Upright view of the abdomen demonstrates midline abdominal/right lower thoracic surgical skin staples (open arrows) along with retained contrast in the intrathoracic stomach and proximal small bowel. Multiple air/fluid levels are also present (small arrowheads) and linear air radiodensities are noted overlying the right upper quadrant (large arrowheads).

Diagnosis: Extensive pneumatosis intestinalis and portal venous air developing after small bowel infarction.

Discussion

Pneumatosis intestinalis (PI) was first described in the 1700s and was initially designated "pneumatosis cystoides intestinorum."¹ PI is simply defined as the presence of gas within the bowel wall and has been described under many names including: pneumatosis cystoides intestinalis, bullous emphysema of the intestine, intestinal gas cysts, and peritoneal lymphopneumatosis.

Detection of PI is usually made by a plain radiograph of the abdomen or by abdominal CT. The clinical context of PI is variable. It can occur as a benign entity in adults with chronic obstructive pulmonary disease (COPD) or as an incidental finding in endoscopic mucosal biopsy tissue.² PI is also associated with more serious conditions such as necrotizing enterocolitis (in newborns) and ischemic bowel (in adults, as in this patient).³

After plain film detection of PI, barium studies are usually confirmatory, demonstrating the air filled lesions protruding into the contrast column. CT can also be used as a confirmatory study to identify gas within the bowel wall.⁴ A bubbly or frothy pattern is sometimes seen in newborns with necrotizing enterocolitis, in adults with bowel infarction, or in emphysematous gastritis caused by a gas-producing infection in the gastric wall.⁵

The linear type of PI, seen as streaks of gas oriented parallel to the bowel wall, can also be seen as either a benign finding or associated with severe illness. Its association with portal venous gas usually means a life-threatening infection or infarction of the gastrointestinal tract. In contrast, rupture of subserosal cysts can result in "benign pneumoperitoneum" or pneumoperitoneum without symptoms.⁶

PI is associated with numerous other intestinal disorders including peptic ulcer disease, intestinal obstruction, and jejunoileal bypass. It can also be associated with other systemic illnesses such as cystic fibrosis, collagen vascular diseases, and acquired immunodeficiency syndrome (AIDS).¹

Commonly proposed mechanisms for many of the above disorders include increased intraluminal gas pressure within the bowel, followed by dissection of gas into the bowel wall, or the presence of gas-forming organisms within the bowel wall itself resulting from a preexisting bowel injury.⁷ Gaseous composition of PI has been demonstrated to be either that of air or of a gas containing high levels of hydrogen thereby supporting two theories for the development of PI.⁸

The mechanical theory proposes that gas dissects into the bowel wall from either the intestinal lumen or the lung. This theory is supported by the occurrence of PI after endoscopy. PI has also been produced experimentally by inflating an

excised cecum in which mucosal incisions have been made and by the injection of air into tissues surrounding the thoracic aorta of cadavers.⁸

The bacterial theory proposes that gas-forming bacteria enter the submucosa through defects within the mucosal lining.⁹ It is thought that hydrogen gas accumulates from the gas-forming bacteria and, under pressure, diffuses into the bowel wall. Pneumatosis is, therefore, the end result of gas dissecting into the intestinal wall; the type of gas and its clinical significance depend on the specific reason for the gaseous distension.

In summary, pneumatosis intestinalis is a rare condition associated with numerous other clinical entities. It is a sign rather than a disease, and the significance of PI must be judged in conjunction with the clinical scenario in order to avoid unnecessary treatment. In the presence of bowel infarction, urgent surgery should remain the treatment of choice, as this condition is usually fatal if not treated expediently.

The patient presented above was taken to the operating room where multiple loops of small bowel were found to be necrotic and discolored. He recovered after a lengthy hospital stay and has resumed a normal diet. He is currently asymptomatic and is scheduled for a follow-up CT scan to exclude recurrent/metastatic disease.

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ONCOLOGY... TODAY

The management of cancer pain

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ABSTRACT

The majority of patients with cancer experience significant pain during their illness. Most cancer pain can be readily managed with oral analgesic therapy. However, cancer pain is often under-treated because of poor communication between physicians and patients and inadequate training of physicians in pain management. A systematic pain-oriented history, pain intensity assessment, physical exam, and diagnostic evaluation are needed to delineate the cause of pain. A therapeutic plan can then be tailored to the patient's needs, preferences, and severity of pain. This paper reviews the evaluation and treatment of cancer pain, with guidelines for initiating and monitoring non-opioid and opioid analgesic therapy.

Introduction

More than 70% of patients with cancer develop significant pain during the course of their illness.¹ Of patients with cancer, 20% to 50% present with pain, 33% have pain during treatment, and 75% to 90% have moderate to severe pain in advanced stages.^{2,3} Despite consensus that 85% of these patients could be well palliated with existing pain treatments, cancer pain management remains poorly managed.⁴ Thus, it is important for all physicians who encounter patients with cancer to learn to elicit and recognize symptoms, evaluate the source of pain, and provide appropriate treatment.

As innovations in cancer therapy prolong survival, the probability increases that patients with cancer will require care from providers other than oncologists. Furthermore, the shift toward managed care favors the evaluation of problems such as pain, at least initially, in a nonspecialist setting. Thus, internists, family practitioners, and other primary care providers are increasingly called upon to provide cancer pain assessment and treatment.

Barriers to pain treatment

The under-treatment of cancer pain, although multifactorial, is often due to

poor physician-patient communication. Patients may be hesitant to report symptoms of pain, either because they assume pain is an inevitable consequence of cancer or they perceive stoicism as an important attribute in the fight against cancer. Patients may also be reluctant to admit they have pain for fear the pain heralds a recurrence or progression of their disease. In addition, patients may be concerned about opiate addiction or toxicity.

Physicians are also responsible for the under-treatment of cancer pain. Many, if not most, lack formal training in pain evaluation and management; this may make physicians wary of eliciting their patients' concerns about pain. Many physicians also have difficulty assessing the intensity of patients' pain. Patients with chronic pain often do not manifest the typical sympathetically-mediated physiologic responses to acute pain, and thus, do not appear to be in pain. This may lead physicians to minimize patient reports of pain intensity. In a study comparing patient and caregiver ratings of cancer pain, there was no correlation between the ratings of patients with severe pain and their providers' estimation of their pain.⁵ In addition, physicians may be reluctant to prescribe opioids, even when appropriate, because of concern about addiction, tolerance, toxicity, regulatory scrutiny, or illicit diversion.

Types of cancer pain

Cancer pain falls into three broad categories, and the type of pain guides the diagnostic evaluation and management.⁶ Nociceptive pain results from the activation of nociceptors in cutaneous and deep tissues. It is typically well localized and is described as 'gnawing' or 'aching'. Examples include bone metastases, incisional pain, and musculoskeletal inflammation. Visceral pain results from infiltration, compression, or distention of the thoracic and abdominal viscera, usually as a result of tumor primary or metastatic growth.

The object of this new series, Oncology Today, is to provide concise review of current oncologic topics for the non-specialist in the field.

Section editor: Ernest C. Borden, M.D., director, Marlene and Stewart Greenebaum Cancer Center (University of Maryland Cancer Center)

This pain is poorly localized and is described as 'deep' or 'squeezing'. Visceral pain may be referred to cutaneous sites remote from the lesion. Nociceptive and visceral pain frequently respond to non-steroidal anti-inflammatory agents (NSAIDs) and opioids. Neuropathic pain results from injury to the peripheral or central nervous system. It is most commonly associated with tumor compression, or infiltration of peripheral nerves or the spinal cord. It is typically described as a burning, tingling discomfort, often with superimposed shock-like paroxysms. Examples include brachial plexopathy, cauda equina compression, and chemotherapy-induced neuropathy. This type of pain does not appear to be as sensitive to opioids as nociceptive pain.

Evaluation of the patient with cancer pain

Prompt and detailed evaluation of the patient with cancer pain is essential to provide relief, target treatment, and preserve function. Approximately 70% of pain in cancer patients results from tumor invasion or compression of soft tissue, bone, or neural structures. Thus, a comprehensive history, physical, and laboratory/radiologic evaluation are essential not only to establish an appropriate treatment for the pain, but to address the underlying cause. For instance, a patient with cancer who presents with back pain may have osteoarthritis, degenerative disk disease, metastatic bone disease, or epidural extension of tumor. While empirical treatment may provide temporary relief for all four types of pain, failure to identify the cause of the back pain in the presence of epidural extension of tumor could lead to compression of the spinal cord, resulting in paralysis and bowel and bladder dysfunction.

1. History

A detailed history should be obtained, with particular attention to all characteristics of the pain: location (of each type of pain), onset, duration, temporal pattern, quality, radiation, association with activity, positional aspects, and therapeutic modalities that have and have not been successful. A detailed medical history may reveal underlying conditions responsible for the pain (e.g., peptic ulcer disease). A detailed oncologic history is also essential to determine if the pain is related to previous or ongoing therapy. Approximately 20% of pain in cancer patients occurs as a result of surgery, chemotherapy, or radiation therapy.²

2. Pain intensity assessment tools

After completing a history, the physician should obtain from the patient an assessment of pain intensity using a visual analog or descriptor scale. Pain is always subjective and is unmeasurable by any neurophysiological or chemical test. Therefore, patient ratings are the

only appropriate measure of pain intensity. **Table 1** reproduces several validated scales.⁴ These scales provide a basis for choosing an appropriate initial treatment and for evaluating the effectiveness of therapy. The goal of therapy is to ensure patient comfort, which requires serial assessment of patient pain intensity ratings.

3. Physical exam

A detailed physical examination, with particular attention to the neurologic exam, is the next essential component.

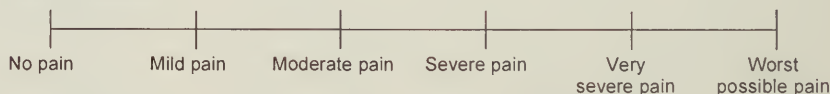
4. Diagnostic evaluation

Following the formulation of a differential diagnosis for the pain, selective diagnostic tests may be indicated, with particular attention to studies that assess the need for glucocorticoids, nerve blocks, chemotherapy, radiation therapy, or surgery. Blood tests, tumor markers, radiographs, computed tomography (CT) scan, magnetic resonance imaging (MRI), bone scan, myelogra-

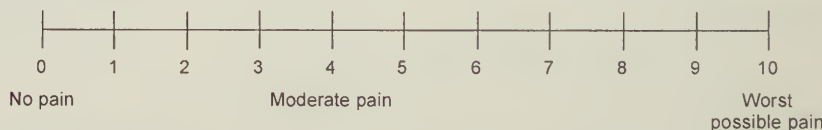
Table 1. Pain intensity assessment tools

PAIN INTENSITY ASSESSMENT TOOLS

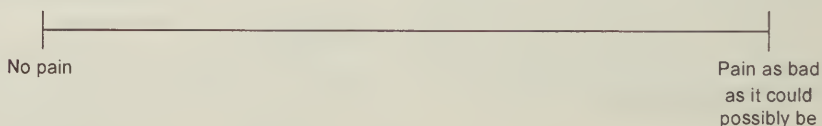
♦ Descriptive scale



♦ Numeric scale



♦ Visual analog scale*



A 10-cm baseline is recommended.

From Grossman SA, Gregory RE: Pain. In *Current Cancer Therapeutics*, 1st ed., Kirkwood JM, Lotze MT, Yasko JM, eds. Philadelphia: Current Medicine, 1994.

phy, lumbar puncture, EMG, and nerve conduction studies are sometimes necessary to delineate the cause of the pain.

Guidelines for treatment

Analgesic therapy should be individualized for each patient. There are several general guidelines for designing an appropriate treatment regimen. First, the regimen should take into account the patient's overall medical and psychosocial status. For instance, issues such as the ability to take oral medications, ability to adhere to a complicated medication regimen, history of substance abuse, degree of social support, and cultural beliefs and practices should be considered in tailoring a regimen to a patient's specific needs.

Second, the regimen should be designed to minimize the burden to the patient, both in terms of cost and complexity. Oral regimens should be tried initially, unless the patient is unable to tolerate oral medications. Generic substitutions should be considered when available. Sustained-release preparations may facilitate compliance.

Third, the physician should continually educate the patient about proper medication administration and possible side effects. The patient on NSAIDs should be advised about the need to discontinue therapy prior to invasive procedures or following symptoms of gastrointestinal intolerance. If opioids are prescribed, the patient should be warned about sedation and nausea. Constipation should be treated prophylactically; almost all patients receiving around-the-clock opioids need regular laxative therapy.⁷

Fourth, following the initiation of any treatment regimen, frequent reassessment is necessary to determine if analgesia is sufficient, if the pain is worsening, or if new pains have arisen. In general, for inadequate pain relief, dose increase or substitution of agents

Table 2. Selected non-opioid analgesics

<u>Drug</u>	<u>Initial dose (mg)</u>	<u>Maximum daily dose (mg)</u>	<u>Comments</u>
♦ Acetaminophen	650 q6h	6000	Not anti-inflammatory. Hepatotoxic at high doses. No effect on platelet aggregation. No GI toxicity.
♦ Aspirin	650 q6h	6000	GI intolerance common. Causes platelet disaggregation.
♦ Ibuprofen	600 q6h	4200	
♦ Naproxen	250 q12h	500	Twice daily dosing.
♦ Indomethacin [†]	25 q8h	200	
♦ Ketorolac	30 q6h	120	Not recommended for prolonged use.
♦ Choline Magnesium Trisalicylate	500 q12h	3000	No effect on platelet aggregation.

within the same analgesic category should be tried before switching to a stronger agent.

Options for treatment

1. Non-opioid analgesics

For the patient with mild to moderate pain, aspirin, acetaminophen, or NSAIDs may provide sufficient relief. Because of their combined analgesic and anti-inflammatory properties, aspirin and NSAIDs are especially effective in treating moderate to severe pain caused by bone metastases, mechanical distention of the periosteum, mechanical compression of muscles and tendons (such as with sarcoma), mechanical distention of the pleura or peritoneum (such as with intrathoracic or intra-abdominal tumors), and inflammation and stiffness of joints or muscles due to anticancer therapy.⁸ Acetaminophen has similar analgesic potency, although it lacks anti-inflammatory activity.

These agents must be given frequently and at regular intervals in order to achieve and sustain a therapeutic effect.⁸ Unlike opioids, tolerance to these agents does not occur, but there is a ceiling of analgesic

effect, beyond which increasing the dose does not produce additional analgesia. Extreme caution must be exercised to avoid complications and/or contraindications such as thrombocytopenia, bleeding, gastric ulceration, renal failure, and hepatic dysfunction.⁹ Table 2 lists appropriate doses and intervals.

2. Weak opioids

For patients with moderate to severe pain, or for patients whose pain is not sufficiently controlled by the non-opioid analgesics, a weak opioid should be given alone or in combination with a non-opioid analgesic. Weak opioids, such as codeine, oxycodone, and hydrocodone are appropriate for moderate pain (Table 3). Agents that combine weak opioids with acetaminophen or NSAIDs are also available. The use of such combination agents can achieve synergistic analgesia and a consequent reduction in dose-related side-effects associated with opioids.¹⁰

3. Strong opioids

For persistent or severe pain, the weak opioid should be replaced by a more potent opioid, such as mor-

phine, hydromorphone, or fentanyl. **Table 3** provides dosing guidelines, although there is not one optimal or maximal dose of strong opioids.⁷ The appropriate dose is that which achieves pain control without respiratory compromise or unwanted sedation. The initial dose should be based on the patient's pain intensity, and subsequent doses should be titrated according to improvement or worsening of pain. Pain can be controlled in most patients with less than 240 mg of daily oral morphine, although some patients require up to 1800 mg of morphine per day.⁷ The appropriate dosing interval is determined by the type of opioid and the route of adminis-

tration. If pain relief does not last throughout the specified dosing interval, the dose should be increased.

In general, full-agonist opioids should be employed in the treatment of cancer pain. Opioids are best given "around the clock" to avoid breakthrough pain. While controlled-release preparations are often convenient and appropriate for long-term administration, immediate release opioid preparations are recommended in the dose titration period.⁶ Transdermal or parenteral routes of administration are often appropriate for patients who cannot tolerate oral medications. Long-term rectal suppository therapy or repeated intramuscular injections are unnecessarily burdensome to patients.

While tumor progression is the most common reason for increasing opioid requirements, tolerance to long-term opioid administration may also necessitate increased doses.

Physicians often confuse the physiologic consequences of long-term opioid administration — tolerance and physical dependence — with psychological dependence and addiction.⁹ This confusion can lead to ineffective administration of opioids and undertreatment of cancer pain. Although often used by both patients and physicians as a reason to avoid the use of opioids, addiction is an extremely rare consequence of opioid administration for cancer pain.^{11,12}

In the patient who has developed opioid dependence, sudden discontinuation or dose reduction will precipitate physiologic withdrawal. Patients with diminishing opioid requirements should receive at least 25% of their previous day's opioid dose to prevent withdrawal.

Mild opioid-induced side effects are usually readily controlled by providing symptomatic therapy

Table 3. Selected opioid analgesics

<u>Drug</u>	<u>Route</u>	<u>Equi-analgesic dose (mg)*</u>	<u>Duration of action</u>	<u>Comments</u>
♦ <i>Weak opioids</i>				
Codeine	PO	200	3-6 hours	Max. dose = 240 mg/day. Not effective in patients with CYP2D6 enzyme deficiency or inhibition.
	IV	130		
Oxycodone	PO	30	3-6 hours	Available alone and in fixed combinations with acetaminophen or aspirin. 12-hour controlled release preparation available.
Hydrocodone	PO	NA	4-6 hours	Only available in fixed combinations with acetaminophen or aspirin.
♦ <i>Strong opioids</i>				
Morphine	PO	30-60	4-6 hours	Equi-analgesic dose of oral morphine varies with the chronicity of morphine exposure: the chronic equi-analgesic dose is 30 mg, whereas the single equi-analgesic dose is 60 mg.
	PO (SR)	60	8-12 hours	
	PO (SR)	60	24 hours	
	IV, IM	10	3-6 hours	
Hydromorphone	PO, PR	7.5	3-4 hours	
	IV, IM	1.5	3-4 hours	
Fentanyl	Transdermal	+	+	Patches available in 25, 50, 75, and 100 mcg per 72-hour dosages.

*..... Approximate potency relative to 10 mg of parenteral morphine.

+..... The microgram-per-hour dose of transdermal fentanyl is equal to one-half of the milligram-per-day dose of oral morphine.

Note: ... Meperidine is not recommended for first-line therapy because of its short half-life and the risk of toxic metabolite accumulation.

Methadone and levorphanol are not recommended for first-line therapy because of their long half-lives and the risk of toxicity.

Table 4. Adjuvant pain medications

<u>Drugs</u>	<u>Initial oral dose (mg)</u>	<u>Indication for use</u>	<u>Comments</u>
♦ Antipsychotics			
Halperidol	2,q6h	Delirium, agitation, neuropathic pain	Little available data of efficacy for pain relief
Chlorpromazine	10,q6h		
♦ Tricyclic antidepressants			
Imipramine	75,qhs	Burning, deafferentation pain, pain complicated by insomnia or depression	Pain relief not seen for 7-10 days after initiating therapy; monitor serum levels
Amitriptyline	75,qhs		
Desipramine	25,tid		
Doxepin	10,tid		
♦ Anticonvulsants			
Carbamazepine	100,bid	Lancinating neuropathic, postherpetic, or phantom limb pain	Monitor serum levels; dose titration may be necessary
Phenytoin	100,tid		
♦ Benzodiazepines			
Clonazepam	0.5,bid	Muscle relaxation, neuropathic pain	Avoid abrupt withdrawal; other benzodiazepines are not indicated for neuropathic pain
♦ Phenothiazines			
Methotrimeprazine	10,q4-6h*	Only methotrimeprazine is an analgesic	Avoid promazine and prochlorperazine – may be antianalgesic
♦ Steroids			
Dexamethasone	20X1, then q6h	Pain due to tumor infiltration of neural structures, bone pain, cord compression	Higher doses may be needed for cord compression

*IV dose recommended since not available in oral formulation.

From Grossman SA, Gregory RE: Pain. In *Current Cancer Therapeutics*, 1st ed., Kirkwood JM, Lotze MT, Yasko JM, eds. Philadelphia: Current Medicine, 1994.

(such as anti-emetics for nausea), by allowing time for tolerance to develop (which often relieves sedation and mild cognitive impairment), or by gradual dose reduction. Life threatening toxicities, such as respiratory compromise or hemodynamic collapse, should be treated with naloxone.

4. Adjuvant drugs

Adjuvant drugs often enhance an analgesic regimen.⁴ Steroids reduce edema associated with brain and epidural metastases. Antidepressants may elevate mood, relieve insomnia, and alleviate neuropathic pain. Anticonvulsants, such as carbamazepine and phenytoin, may be effective against neuropathic pain, such as trigeminal or postherpetic neuralgia or plexopathy. Amphetamines may decrease

opioid-induced sedation. **Table 4** provides dosing guidelines for adjuvant pain medications.⁴

When to refer

In most instances, cancer pain can be well managed by a primary care provider. However, several conditions warrant timely referral to a specialist. Pain due to tumor growth or extension may require immediate tumor reduction therapy, not only to alleviate pain, but to prevent irreversible neurologic or visceral sequelae. Any patient whose pain might be alleviated by tumor reduction therapy should be referred to the appropriate oncologist, radiation therapist, or surgeon. The urgency of such a referral is often determined by the underlying cause of the pain. Certainly, emergent radiation therapy is indicated for pain secondary to epidural

extension of tumor or metastases to weight-bearing bones. Likewise, pain secondary to intestinal obstruction warrants emergent surgical evaluation. Cancer patients with rapidly escalating pain, for whom the cause of pain has not been determined, should be evaluated by an oncologist. Patients who may benefit from regional anesthesia techniques (such as local anesthetic blocks, neurolytic blocks, and epidural/intrathecal opioid administration) also require specialist evaluation.

Conclusion

Cancer pain can be evaluated and alleviated effectively in most patients using the simple guidelines outlined above. Effective communication between physicians and patients is the first and most important step in pain management. Patients with cancer

should be asked about pain symptoms at every visit and reassured that pain control is an important and achievable goal. The patient's pain rating, using a validated pain intensity scale, should be obtained at each visit and incorporated into the physician's evaluation and plan. Any patient with pain should have a history, physical exam, and diagnostic evaluation directed toward determining the source of pain. Tumor reduction therapy may be needed to alleviate the pain, requiring referral to an oncologist, radiation therapist, or surgeon. However, if the pain cannot be controlled through such measures, a pharmacologic approach is indicated.

Although many pharmacologic options exist for pain control, a limited drug repertoire is usually sufficient to manage pain symptoms. Each physician should choose several non-opioid and opioid analgesics (with a range of potencies), and then gain experience with the pharmacologic characteristics of these agents, including dose adjustment, efficacy, and toxicity.

A treatment regimen can then be tailored to the patient's individual needs and preferences. Depending on pain severity, non-opioid analgesics or opioids may be the appropriate initial agent. Any patient on an analgesic regimen requires frequent reassessment to titrate the medication, avoid toxicities, and evaluate any new or uncontrolled pain.

Pain is the most common symptom that brings a patient to a physician's attention.¹³ All clinicians, therefore, need to be comfortable with the evaluation and management of pain. This is particularly true for clinicians who care for patients with cancer, as pain is one of the most common and feared symptoms of cancer. Through regular communication with patients, careful evalu-

ation of signs and symptoms of pain, and mastery of a few analgesic agents, clinicians can alleviate both the fear and symptoms of cancer pain.

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Alternative Medicine Digest, Issue 16

FROM THE BPQA

■ School sports exams: the case of Dr. D

An orthopedic surgeon had been doing sports physicals at a local school for many years. On the day in question, he arrived and found that the woman who usually assisted him would not be available. Since many students had already signed up, he proceeded without her assistance. The only available room was an office with windows to a corridor. To improve the students' privacy, he did not use the overhead lights. He examined about 15 teenagers, both boys and girls. He conducted the exams with the students seated on a desk, and he examined the breasts of several girls.

Subsequently, the girls discussed their examinations with one another. One girl learned that not all of the girls had been given breast examinations. The perception was that only attractive girls who were not accompanied by a parent had been given breast examinations. A parent was consulted. The Department of Social Services was contacted and the police were called. A report was made to the Board of Physician Quality Assurance (BPQA).

BPQA investigators interviewed several of the girls. The girls related that they had mutely submitted to breast examinations as part of their sports physicals. One girl alleged that it was very unlike the examination her gynecologist does; she described having both breasts cupped and massaged simultaneously by the orthopedist. None of the

girls said they had voiced any concerns about their breasts which should have precipitated an examination. Emotions were running high and the girls' parents were outraged.

The BPQA voted its intent to summarily suspend the physician and he was given an opportunity to have a BPQA hearing prior to suspension. At the hearing, the physician described his typical physical examination as taking 5 to 10 minutes. He claimed he did breast examinations on the girls who either had a breast complaint or who were on birth control pills. He couldn't remember which girls had breast examinations, and he didn't record a breast examination on any of the forms. He related that he was stunned that the girls had complained as none of them voiced any objection during the examinations.

■ What would you do if you were on the licensing board and were faced with this set of circumstances? Is summary suspension warranted to protect public safety? Is sufficient information available at this point to render a fair decision?

*Comment by Cheryl Winchell, M.D.,
Secretary/Treasurer, BPQA*

This case illustrates at least three important issues. First, physicians should realize that when they conduct an examination outside of their regular offices, many of the signals that reinforce the professional nature of

the interchange may be lost. An adequate examination room with appropriate lighting, a drape for the patient, and the presence of other professionals all reassure a patient that a physician's examination is routine and appropriate. Second, although many patients are comfortable without a chaperon, those most likely to wish a chaperon are teenage girls undergoing an intimate examination by a male physician, particularly if they don't have an ongoing relationship with the doctor. Third, although the physician thought he had the girls' consent to examine their breasts as part of their sports physicals, the girls mutely submitted, even though some of them were mortified and had no idea that a breast examination would be performed that day. In the situation described, clear consent and clear purpose for this intimate examination was not at all clear.

■ Action taken by the BPQA

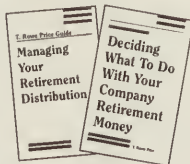
The BPQA decided not to summarily suspend the physician, allowing him to continue in practice until he had a full hearing before an administrative law judge. He was asked not to perform any more school sports physicals in the interim. The administrative law judge heard the case. Several of the students testified. The physician testified and called his own witnesses, including some of the other female students he had examined that same day. After a full hearing, the administrative law judge held that the BPQA had not met the burden of proof by "clear and convincing evidence" that the physician had acted in an unprofessional manner. Subsequently, the BPQA dismissed the charges. ■

The opinions expressed in this column are those of Dr. Winchell and are not endorsed by the BPQA.

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DHMH



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Ebenezer Israel, M.D., M.P.H., Director
Epidemiology & Disease Control Program

EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

March 1997

Reporting Reminder, Emerging Infections Program Grant, Correction on Standard Precaution Summary, Syphilis Update, and Influenza Season Update

Report Communicable Disease Cases to Your Local Health Department

Recent cases of hepatitis A have been reported more than two weeks after onset. They have illustrated the importance of rapid case diagnosis and reporting. Once reported, the local health departments investigate the cases to determine their **source** and potential **spread** through food handling, patient, or child care, and intervenes to stop transmission. **Delayed reporting leads to additional spread. Report quickly to your LHD.**

Maryland Receives Emerging Infection Grant from CDC

Maryland is one of eight states to be funded as an Emerging Infection Program state. The Maryland Department of Health and Mental Hygiene will work in conjunction with the Johns Hopkins School of Hygiene and Public Health and the University of Maryland at Baltimore to enhance surveillance for bacterial invasive diseases and foodborne pathogens.

Notice of Revision

Please note that a revision has been made to the **Summary of the Guidelines for Isolation Precautions in Hospitals: HICPAC Recommendations for Isolation Precautions in Hospitals**, printed in the January 1997 issue of the *Maryland Medical Journal*, p. 44. A corrected version of this summary can be found on the following page.

In our previous summary, *Streptococcus pneumoniae* pneumonia was listed as an example disease under the column labeled Droplet Precautions. ***S. pneumoniae* pneumonia does not require Droplet Precautions.** Rather, Standard Precautions are necessary for this illness.

If you have any questions or concerns regarding this revision, please contact the Maryland Department of Health and Mental Hygiene, Epidemiology and Disease Control Program at (410) 767-6677.

Summary of the Guidelines for Isolation Precautions in Hospitals: HICPAC Recommendations for Isolation Precautions in Hospitals (*Infection Control and Hospital Epidemiology* 1996,17(1):53-80) - REVISED JANUARY 1997

Standard Precautions Summary

Standard Precautions are designed to incorporate the protection against blood-borne pathogens achieved by **Universal Precautions**, and the protection against other pathogens achieved by **Body Substance Isolation**. Standard Precautions are to be used on **ALL** hospital patients, regardless of their diagnosis or presumed infectious status, when coming into contact (or risk of contact) with any of the following: (1) **blood**, (2) **all body fluids, secretions and excretions except sweat**, (3) **nonintact skin**, or (4) **mucous membranes**.

Standard Precautions consist of the following nine components:

- (1) Routine hand washing
- (2) Consistent and correct glove use (i.e., glove changes with hand washing between patients)
- (3) Appropriate use of masks, eye protection, and face shields
- (4) Appropriate use of gowns [when necessary]
- (5) Routine cleaning or disposal of patient-care equipment
- (6) Regular cleaning of all environmental surfaces
- (7) Appropriate handling of contaminated linen
- (8) Strict adherence to occupational safety requirements
- (9) Effective management of patients with poor hygienic behaviors



Transmission-based Precautions Summary

Transmission-based precautions consist of **additional** measures designed to be used in addition to Standard Precautions to further reduce the risk of disease transmission. Transmission-based precautions are divided into the three categories listed below. Specific use of a category of transmission-based precautions is based upon the disease(s) of the patient. A partial list of disease examples are listed below; **for a complete list of diseases please refer to *Infection Control and Hospital Epidemiology* 1996,17(1):53-80, Appendix A.**



Airborne Precautions

- (1) Place patient in a private room or cohort.
- (2) Use respiratory protection when appropriate.
- (3) Limit patient transport within the facility.
- (4) Use additional precautions with tuberculosis.*

Example Diseases:

TB-pulmonary or laryngeal
Measles
Chickenpox



Droplet Precautions

- (1) Place patient in a private room or cohort; when not possible, maximize distance between patients.
- (2) Wear mask when working closely with the patient.
- (3) Limit patient transport within the facility.

Example Diseases:

H. influenzae meningitis
N. meningitidis meningitis
Diphtheria
Pertussis
Influenza



Contact Precautions

- (1) Place patient in a private room or cohort; when not possible, consult the infection control practitioner (ICP).
- (2) Wear gloves upon entrance to room and at all times.
- (3) Wash hands with antimicrobial soap upon leaving the room taking care not to touch environmental surfaces.
- (4) Wear a gown when entering the room if contamination is possible.
- (5) Limit patient transport within the facility.
- (6) Dedicate the use of personal, noncritical medical equipment to a single patient.
- (7) Use additional precautions for preventing the spread of vancomycin resistance.**

Example Diseases:

MRSA/ VRE infection or colonization**, *C. difficile* with diarrhea, Shigellosis if diapered or incontinent, Scabies

*CDC. Guidelines for preventing the spread of tuberculosis in health care facilities. MMWR. 1994;43(RR-13):1-132.

**HICPAC. Recommendations for preventing the spread of vancomycin resistance. AJIC 1995;16:105-13.

Syphilis Cases Remain High in Baltimore

Infectious and congenital syphilis cases remain high throughout 1996 for Baltimore City and Baltimore County. Provisional data indicate that Baltimore City will likely report 523 cases of primary and secondary syphilis for 1996. This figure is 49% above the 351 cases reported in 1995, and 172% above the 192 cases reported in 1994 (figure 1). From 1989 through 1994, Baltimore City reported an average of 191 cases of infectious syphilis per year. An alarming result of the increase in adult cases has been an increased number of congenital syphilis cases. Provisional data indicate that 37 cases of congenital syphilis have been reported for 1996-- **eight of these infants were stillborn**. Baltimore City reported an average of 12 cases per year from 1993 through 1995.

An increase in reported cases in Baltimore County was not seen until early 1996. Provisional reports indicate that the County will likely see 56 cases of primary and secondary syphilis in 1996, an **increase of 250% from the 16 reported in 1995**. Though relatively small in actual number, the magnitude of the increase is alarming. Congenital syphilis has increased as well. Five cases, of which two were stillborn, have been reported for 1996 compared to no cases in 1995.

We urge every physician in the Baltimore Metropolitan Area to assist us in reversing these

alarming trends. Early and appropriate screening, along with rapid diagnosis, treatment, and epidemiologic follow-up are crucial steps in successful syphilis control and congenital syphilis prevention.

✓ **Know the symptoms of syphilis.** These include lesions in the urogenital or oral areas; generalized body rashes or rashes on palms, soles, or face; moist papular lesions in the urogenital or perianal area; lymphadenopathy; alopecia.

✓ **Test symptomatic and high risk asymptomatic patients for syphilis.** Identified risks include a history of multiple sex partners, unprotected sex, infection with other STDs, and use of illicit drugs, especially crack cocaine.

✓ **Test all pregnant women at first prenatal visit and at 28 weeks.**

✓ **Do a STAT serologic test for syphilis on all women presenting in labor who have a history of unknown, spotty, or no prenatal care.** Neither infant nor mother should be discharged before results are known.

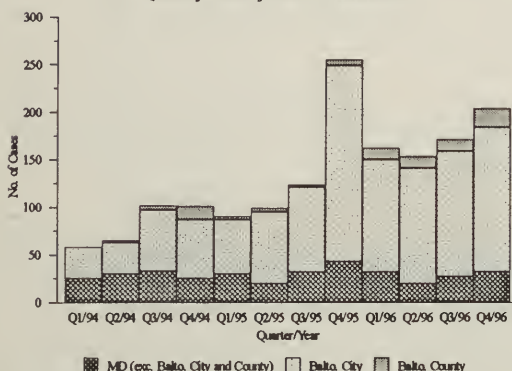
✓ **Treat all diagnosed syphilis patients according to CDC 1993 Treatment Guidelines.**

✓ **Report, immediately, all cases of primary and secondary syphilis and all cases of syphilis in pregnant women to local health officials so that rapid follow-up may take place.** Telephone reporting is encouraged during this epidemic.

✓ **Consult with the local health department on issues related to diagnosis, treatment, patient follow-up, and partner referral.**

Obtain assistance and more information by contacting the STD Programs in Baltimore City (396-4448; fax 625-0688), Baltimore County (887-2713; fax 828-0896), or Maryland Department of Health and Mental Hygiene (767-6688; fax 333-5529).

Primary and Secondary Syphilis, Maryland 1994-1996
Quarterly Cases by Selected Jurisdictions



Source: Epidemiology & Disease Control, DHMH, 1997

Maryland Hit Hard During 1996-97 Influenza Season

Maryland has had a significant increase in the incidence of influenza and influenza-like illness (ILI) during the 1996-97 influenza season. During the 1995-96 season, only 58 influenza isolates (56 influenza A, 2 influenza B) were reported to the Maryland Department of Health and Mental Hygiene, Epidemiology and Disease Control Program. However, as of January 28, 1997, a total of 227 isolates (222 influenza A, 5 influenza B) have been reported statewide during this ongoing influenza season.

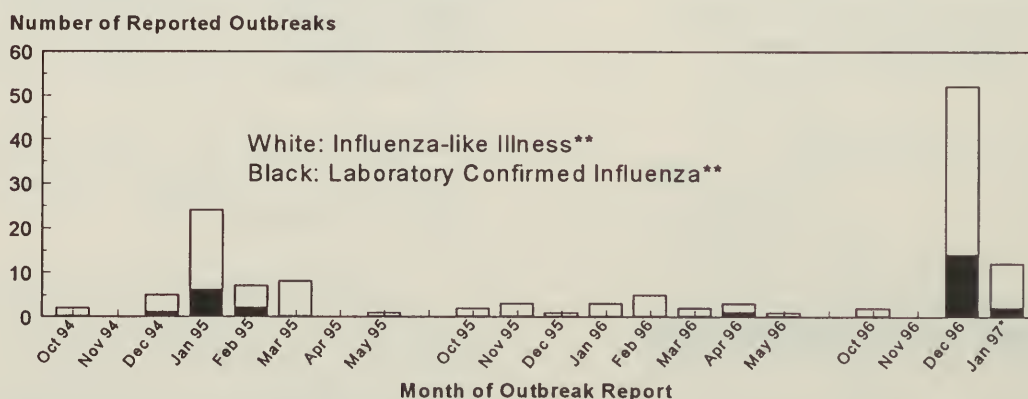
Correspondingly, Maryland's long-term care facilities (LTCF) have also reported a dramatic increase in the occurrence of influenza or ILI outbreaks. As of January 28, 1997, LTCF in Maryland have reported 16 laboratory confirmed influenza A outbreaks and 50 ILI outbreaks. Many of the 50 ILI outbreaks have laboratory results pending and, as a result, may later be characterized as influenza A. During the 1995-96 season, only 1 influenza B and 19 ILI outbreaks were reported. The figure below graphically illustrates the increase in influenza during the 1996-97 season compared to the 1995-96 and 1994-95 influenza seasons.

The increase in influenza activity has been seen nationally as well. For example, for the week ending January 18, 1997, widespread influenza activity was seen in 14 states, and regional activity was seen in 17 states, including Maryland. Additionally, all 3960 isolates of influenza A subtyped by the U.S. World Health Organization have been H3N2. Similarly, all 70 influenza A(H3N2) isolates antigenically characterized by the Centers for Disease Control and Prevention were closely related to A/Wuhan/359/95 and A/Nanchang/933/94, two of the viruses used in the manufacture of U.S. influenza vaccine for the 1996-97 season.

It is important to note that laboratory confirmed influenza is not a reportable disease in Maryland. Reports are collected through active surveillance of several laboratories reporting on a voluntary basis, as well as unsolicited reports. As a result, the true incidence of influenza in Maryland is undoubtedly underestimated.

For further information regarding influenza or to request the DHMH *Guidelines for Influenza and Influenza-like Illness (ILI) Outbreak Investigations in Long Term Care Facilities (9/96)* please call the Epidemiology and Disease Control Program at (410) 767-6677.

Reported Outbreaks of Influenza and Influenza-like Illness in Long Term Care Facilities in Maryland, 1994-95, 1995-96, and 1996-97* Seasons



*Data for January includes only those reports received prior to January 28, 1997; the influenza season is defined as October through May.

** Lab confirmed influenza outbreak: a minimum of one laboratory confirmed case of influenza in the outbreak; Influenza-like illness outbreak: Outbreak of symptoms consistent with influenza but no laboratory confirmed influenza cases (culture results negative, not done, or pending).

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

MGAIMGS series, Department of Mental Hygiene, The Johns Hopkins University, School of Hygiene and Public Health, Keswick Nursing Center, 40th Street, 6:00 p.m. -7:00 p.m., light refreshments served at 5:30 p.m. Credits: TBA. Sponsored by the Maryland Chapter of the American Geriatrics Society (AGS) and the Maryland Gerontological Association (MGA), with assistance of the Geriatrics Committee of the Maryland Academy of Family Physicians. Info: Donna Meisel Weinreich, 410-675-3244 (e-mail: dmeisel@umabnet.ab.umd.edu) or Joseph J. Gallo, M.D., M.P.H., 410-955-0599 (e-mail: jgallo@welchlink.wlech.jhu.edu).

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| Pain management in the older adult | Apr. 29 |
| What's new in the "memory wars": implications for clinical practice , sponsored by the Department of Psychiatry and Behavioral Sciences and False Memory Syndrome Foundation. Credits: 8 Cat 1 AMA credits. Fee: \$175. | Mar. 21 |
| Second annual cardiovascular symposium with the experts , sponsored by the Department of Medicine at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 11.5 Cat 1 AMA credits. Fee: \$175 by March 30; \$225 after March 30. | Mar. 30-31 |
| Diagnosis and treatment of neoplastic disorders , sponsored by The Johns Hopkins Oncology Center. Credits: 14 Cat 1 AMA credits. Fee: \$300/Advanced registration (before Feb. 1); \$325 (Postmarked Feb. 1 and after); \$150/residents, fellows, allied health professionals. | Apr. 3-4 |
| Nuclear oncology: from genotype to patient care , sponsored by The Johns Hopkins University School of Medicine. Credits: up to 18 Cat 1 AMA credits. Fee: \$495/physicians; \$395/residents, fellows, allied health professionals. | Apr. 7-9 |
| Update on Alzheimer's disease and other dementias , Renaissance Harborplace Hotel, Baltimore, MD. Credits: up to 7 Cat 1 AMA credits. Fee: \$145/physicians; \$110/psychologists in practice; \$90/residents, fellows, allied health professionals. | Apr. 12 |
| 25th annual pediatric trends , Johns Hopkins Medical Institute, Department of Pediatrics. This course provides a comprehensive update on new developments of interest to practitioners who care for infants, children, and adolescents. Credits: 42.5 Cat 1 AMA credits; 45.5 AAP credits; 37.5 AAFP prescribed hours. | Apr. 14-19 |
| 38th annual postgraduate institute for pathologists in clinical cytopathology , sponsored by The Johns Hopkins University School of Medicine. Credits: 94.5 Cat 1 AMA credits plus up to 10 hrs. video instruction. Fee: \$2450/physicians; \$1300/senior residents. | |
| Course A (Home study) | Mar.-Apr. |
| Course B , concentrated lecture and laboratory studies, Johns Hopkins Medical Institutions, Baltimore, MD. | Apr. 14-25 |
| Seventh annual clinical care of the patient with HIV infection , sponsored by the Department of Medicine at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$190/residents, fellows, allied health professionals. | Apr. 17-18 |
| 11th annual mood disorders symposium , sponsored by The Johns Hopkins Affective Disorders Clinic, and DRADA. Credit: Cat 1 AMA credit; Cat A credit, Md. State Board of Examiners of Psychologists; Md. State Board of Examiners for Social Workers. Fee: \$50/DRADA members; \$60/other attendees. | Apr. 30 |
| Institute on ministry with the sick , sponsored by the Johns Hopkins University School of Medicine. Credits: 14 Cat 1 AMA credits. Fee: \$150. | May 5-7 |
| Pediatric allergy and immunology for the practitioner , sponsored by the Division of Pediatric Allergy and Immunology. Credits: up to 14 Cat 1 AMA credits. Fee: If postmarked by | May 8-9 |

The Johns Hopkins Medical Institutions (continued)

April 1, \$275/physicians; \$200/residents, other allied health professionals. If postmarked after April 1, \$295/physicians; \$220/residents, other allied health professionals.

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| Critical issues in surgical pathology , sponsored by the Department of Pathology. Credits: 14 Cat 1 AMA credits. Fee: \$400/physicians; \$200/residents, fellows, students. | May 9–10 |
| 42nd annual topics in clinical medicine , sponsored by the Department of Medicine. Credits: 39 Cat 1 AMA credits. Fee: \$750/physicians; \$600/residents, fellows, other professionals. | May 12–16 |

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Self-Directed CME Activities

- Disease management of lipid disorders (audio tape and test).** Credit: 1 Cat 2 AMA credit. Expires 6/97.
- CD-ROM based interactive multimedia radiology teaching file for Mac or PC w/single user licenses (SUL), site licenses (SL), or multisite licenses (MSL).** Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.
- Academic rounds and conference.** Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Office of Continuing Medical Education, Washington University School of Medicine, Campus Box 8063, 660 South Euclid Ave., St. Louis, MO 63110-1093. Unless otherwise noted, seminars will be held at the Washington University Medical Center, Eric P. Newman Education Center (EPNEC), 320 S. Euclid Ave., St. Louis, MO 63110. Info: Cathy Sweeney, 800-325-9862, Fax 314-362-1087.

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| Internal medicine review , Monday evenings, The Jewish Hospital. | Mar.–May |
| Integrated care of the thoracic surgery patient: a seminar for allied health professionals. | Mar. 21–22 |
| Leonard Berg symposium on alzheimer's disease | Apr. 4 |
| Clinical pulmonary update. | Apr. 4–5 |
| Refresher course & update in general surgery , The Ritz-Carlton Hotel, St. Louis, MO. | Apr. 10–12 |

Miscellaneous (continued)

- Delmarva Foundation for Medical Care**, Easton, MD. Credits: 3 Cat 1 AMA credits. Info: Roxanne Rodgers, 410-822-0697.
Health care improvement for physicians Mar. 15
- Eastern wisdom and the practice of psychotherapy**, conference at The Conference Center at Sheppard Pratt, Baltimore, Maryland. Info: Barbara Johnson, Professional Education Programs, Sheppard Pratt Health System, 410-938-4598 (e-mail: riamy@capcon.net). Mar. 22
- 1997 annual session, American College of Physicians**, at the Pennsylvania Convention Center, Philadelphia. The largest meeting for internal medicine and its subspecialties. CME credit available. Pre-session courses March 20-21. Info: 800-523-1546, ext. 2600. Mar. 22-25
- NIH consensus development conference on management of hepatitis C**, Natcher Conference Center, National Institutes of Health, Bethesda, Maryland. Credits: up to 15 Cat 1 AMA credits. Fee: none; early registration encouraged. Contact: Rose Salton, 301-770-3153, Fax 301-468-2245 (e-mail: confdept@tech-res.com). Mar. 24-26
- Problem solving in diagnostic radiology**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Palm Beach, Florida. Credits: 30 Cat 1 AMA credits. Fee: \$694/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). Mar. 29
- 17th annual resident's radiology review course**, sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, California. Designed for senior radiology residents and practicing radiologists. Course covers all major modalities. Credits: 41 Cat 1 AMA credits. Fee: \$695/physicians; \$450/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-8959 (e-mail: webmaster@ryalsmeet.com). Mar. 30-Apr. 4
- Reimbursement and managed care: essential reimbursement strategies in emergency medicine**, Hyatt Regency, Baltimore, Maryland. Sponsor: the American College of Emergency Physicians (ACEP). Credits: 15 Cat 1 AMA credits; 15 Cat 1 ACEP credits. Info: 800-798-1822. Apr. 3-4
- Breast imaging and interventions**, sponsored by The University of California, San Diego, School of Medicine, Department of Radiology, at The Hotel Del Coronado, Coronado, California. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$250/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). Apr. 4-6
- Getting control: effective procedure coding for emergency medicine**, Hyatt Regency, Baltimore, Maryland. Sponsor: the American College of Emergency Physicians (ACEP). Credits: 15.5 Cat 1 AMA credits; 15.5 Cat 1 ACEP credits. Info: 800-798-1822. Apr. 4-6
- Leadership conference**, Stouffer Mayflower Hotel, Washington, DC. Sponsor: American College of Emergency Physicians (ACEP). Credits: TBA. Info: 800-798-1822. Apr. 6-7
- Legislative issues forum**, Stouffer Mayflower Hotel, Washington, DC. Sponsor: the American College of Emergency Physicians (ACEP). Credits: TBA. Info: 800-798-1822. Apr. 7-9
- Building a multidiscipline team for the diagnosis and management of breast disease**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Plaza Hotel, New York, NY. Credits: 21 Cat 1 AMA credits. Fee: \$595/hospital team (no fee for every fifth member — must register together to qualify). Info: Ryals & Associates, 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). Apr. 10-13
- Infectious disease '97: a comprehensive review for the practicing physician**, sponsored by The Center for Bio-Medical Communication, Inc. (CBC), at Renaissance Washington, D.C. Hotel. Credits: 18.25 Cat 1 AMA credits; 18.25 AAFP credits. Fee: by Feb. 3, 1997, \$495/physicians; \$350/physicians-in-training, other allied health professionals. Info: 201-385-8080, Fax 201-385-5650 (e-mail: webmaster@ryalsmeet.com). Apr. 11-13

Miscellaneous (continued)

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| Sixth annual meeting and clinical congress of the American Association of Clinical Endocrinologists (AACE) , Marriott, Philadelphia, Pennsylvania. Credits: up to 36.5 Cat I AMA credits. Info: 904-353-7878. | Apr. 16–20 |
| Problem solving in imaging of the brain, spine, and head and neck , sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Amelia Island, Florida. There will be both didactic lectures and workshops. Credits: 26 Cat I AMA credits. Fee: \$650/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Apr. 17–20 |
| Expanding the cardiac continuum , presented by the Heart Institute at St. Joseph Medical Center at the Hunt Valley Marriott. CME credits available. Fee: \$100/physicians; \$80/nurses, other healthcare professionals. Info: 410-337-1309. | Apr. 18 |
| 2nd annual angio/interventional review course , sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Credits: 10 Cat I AMA credits. Fee: \$215/physicians; \$155/residents, fellows, full-time military, U of F radiology alumni (\$135 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | April 19–20 |
| 9th annual radiology review course: "what you need to know," sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Attendees will improve their knowledge of differential diagnosis, imaging patterns, and techniques of examination. Credits: 50 Cat I AMA credits. Fee: \$695/physicians; \$525/residents, fellows, full-time military, U of F radiology alumni, (\$475 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 701-552-9859 (e-mail: webmaster@ryalsmeet.com). | Apr. 20–25 |
| Adult immunization: strategies that work , presented via satellite by the Centers for Disease Control and hosted by the Maryland Department of Health & Mental Hygiene. Fee: none. Info: Sandra Kash, 410-767-6679. | Apr. 24 |
| Critical care medicine '97: 11th annual review and update , sponsored by the Center for Bio-Medical Communication, Inc., Hyatt Regency, Washington, D.C. Credits: 41.25 Cat I AMA credits, 41.25 AAFP. Fee: By Mar. 21, 1997, \$795/physicians; \$575/physicians-in-training, allied professionals. Info: 201-385-8080, Fax 201-385-5640 (e-mail: cbcbiomed@aol.com). | Apr. 30–May 4 |
| 2nd annual mammography review course , sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Course is designed as an overview of the practical aspects of breast imaging, including interventional procedures. Credits: 15 Cat I AMA credits. Fee: \$295/physicians; \$215/residents, fellows, full-time military, U of F Radiology Alumni (\$185 each, two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Apr. 25–27 |
| Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Shady Grove Adventist Hospital, Montgomery County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. | May 2 |
| Clinical auscultation of the heart , Georgetown University Medical Center, Washington, D.C. Sponsored by the American College of Cardiology. Credits: 21 Cat I AMA credits. Contact: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, MD 20814-1699, 800-253-4636, ext. 695, Fax 301-897-9745. | May 7–9 |
| 56th annual American occupational health conference: discover the reality , sponsored by the American College of Occupational and Environmental Medicine (ACOEM) in conjunction with American Occupational Health Conference (AOHC). Orange County Convention Center, Orlando, Florida. 39 concurrent scientific sessions, 42 postgradu- | May 9–16 |

Miscellaneous (continued)

ate seminars, and 7 two-day training courses. Contact: Kay Cone, ACOEM, 55 W. Seegers Rd., Arlington Heights, IL 60005, 847-228-6850, ext. 152, Fax 847-228-1856.

Cancer prevention in community practice, sponsored by the Medical and Chirurgical Faculty of Maryland, at Physician Memorial Hospital at Hamilton Center, Charles County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **May 13**

Cutaneous melanoma '97: a clinical symposium for primary care practitioners, sponsored by The Skin Cancer Foundation and Memorial Sloan-Kettering Cancer Center, Memorial Sloan-Kettering Cancer Center, New York, NY. Credits: 6.5 Cat 1 AMA credits. Info: Ludmilla Popoff, 212-639-6754. **May 16**

2nd Annual mammography — practical challenges of the '90's, sponsored by X-Ray Associates of New Mexico, P.C., at The Eldorado Hotel, Sante Fe, New Mexico. Credits: 20 Cat 1 AMA credits. Fee: \$650/physicians; \$450/residents, fellows; \$350/technologists, nurses. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **May 23-26**

Self-Directed CME Activities

Maryland physicians' campaign against family violence, module one: domestic violence, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Maryland physicians' campaign against family violence, module two: child maltreatment, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

Fluorescein angiography conference, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00 a.m. – 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.

Sinai Hospital of Baltimore medical grand rounds, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.



PHYSICIAN'S RECOGNITION AWARD

During November 1996, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

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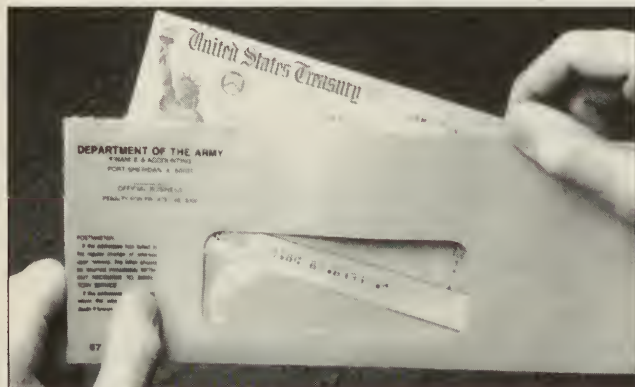
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▼ **What do patients want when physicians make mistakes?** According to a recently published study, patients want honesty. The study reports results of a survey assessing patients' attitudes of physician disclosure and nondisclosure of minor, moderate, and severe mistakes (*Arch Intern Med* 1996;156:2565-2569). Survey respondents indicated that they prefer to know even when mistakes are minor. **Patients reported that they were more likely to consider a law suit if the physician failed to disclose a mistake, and they instead found out by other means (12% if informed versus 20% if not informed).** Also, patients reported an increasing desire to switch physicians for an increase in the severity of the mistake.

▼ **Large-scale studies have demonstrated the effectiveness of thrombolytic agents in reducing mortality in patients who present with myocardial infarction.** It has been reported that many patients who could benefit from thrombolysis are unnecessarily excluded from treatment. **All patients who have appropriate electrocardiographic changes and present within 12 hours of the onset of acute myocardial infarction, regardless of age and history of previous myocardial infarction, should be considered for thrombolytic therapy** (*Am Fam Phys* 1996;54(6):2041-2048).

▼ **In another study regarding thrombolytic administration in patients with suspected myocardial infarction (MI), investigators compared the outcomes for patients with confirmed MI and those for whom MI was ruled out** (*Ann Emerg Med* 1996;28:289-293). **The authors concluded that for 35 patients treated with thrombolytic therapy before MI was ruled out, complications and prognosis were equal to those with MI who received thrombolytic therapy.**

▼ **Baltimore makes a top ten list it is not proud of.** Metropolitan areas with the 10 highest acquired immunodeficiency syndrome (AIDS) annual rates per 100,000 population in the United States for July 1995 to June 1996 are New York, NY (132.0), Miami, FL (117.3), Jersey City, NJ (115.2), San Francisco, CA (109.8), Fort Lauderdale, FL (86.7), West Palm Beach, FL (86.4), Newark, NJ (79.1), San Juan, Puerto Rico (67.0), Baltimore, MD (61.5), and New Orleans, LA (55.8) (*MMWR Morb Mortal Wkly Rep* 1996;45:926-927).

▼ **Because the median survival of patients with multiple myeloma after conventional chemotherapy is three years or less, the authors of a recently published study randomly assigned 200 previously untreated patients under 65 years of age to either the conventional approach or to high-dose therapy and autologous bone marrow transplantation** (*N Engl J Med* 1996;335(2):91-97). **The high-dose bone marrow transplant group had a positive response rate of 81% (including 22% complete responses and 16% very good partial responses). Comparable responses of those treated by conventional therapy was 57% (only 5% complete and 9% very good responses). They, therefore, concluded that high-dose therapy combined with transplantation improved response rate, event-free survival, and overall survival in patients with multiple myeloma.**

▼ **A study designed to understand the reasons for a low percentage of estrogen use among older women found that in order to achieve the benefits of osteoporosis prevention and reduced cardiovascular disease rates, there needs to be increased education of both women and health practitioners** (*Arch Intern Med* 1996;156(6):1293-1297). **The study included nonblack women 65 years of age and older. Of the over 7000 questioned, 17.4% were currently using oral estrogens and 27.2% were past users. Of the past users, approximately 30% discontinued the medication because they felt they no longer needed it and 16.4% because of undesirable side effects, the most frequent being bleeding. Of those women who never started estrogen therapy, the major reasons given were fear that the medication was harmful and that they didn't feel they needed it.**



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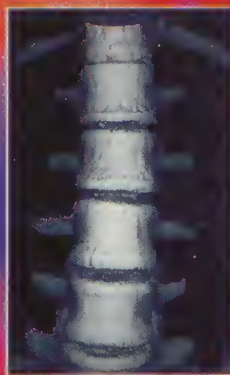
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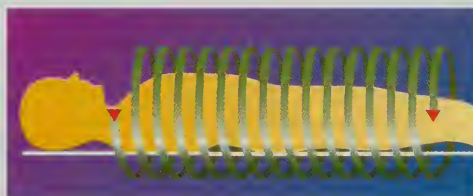
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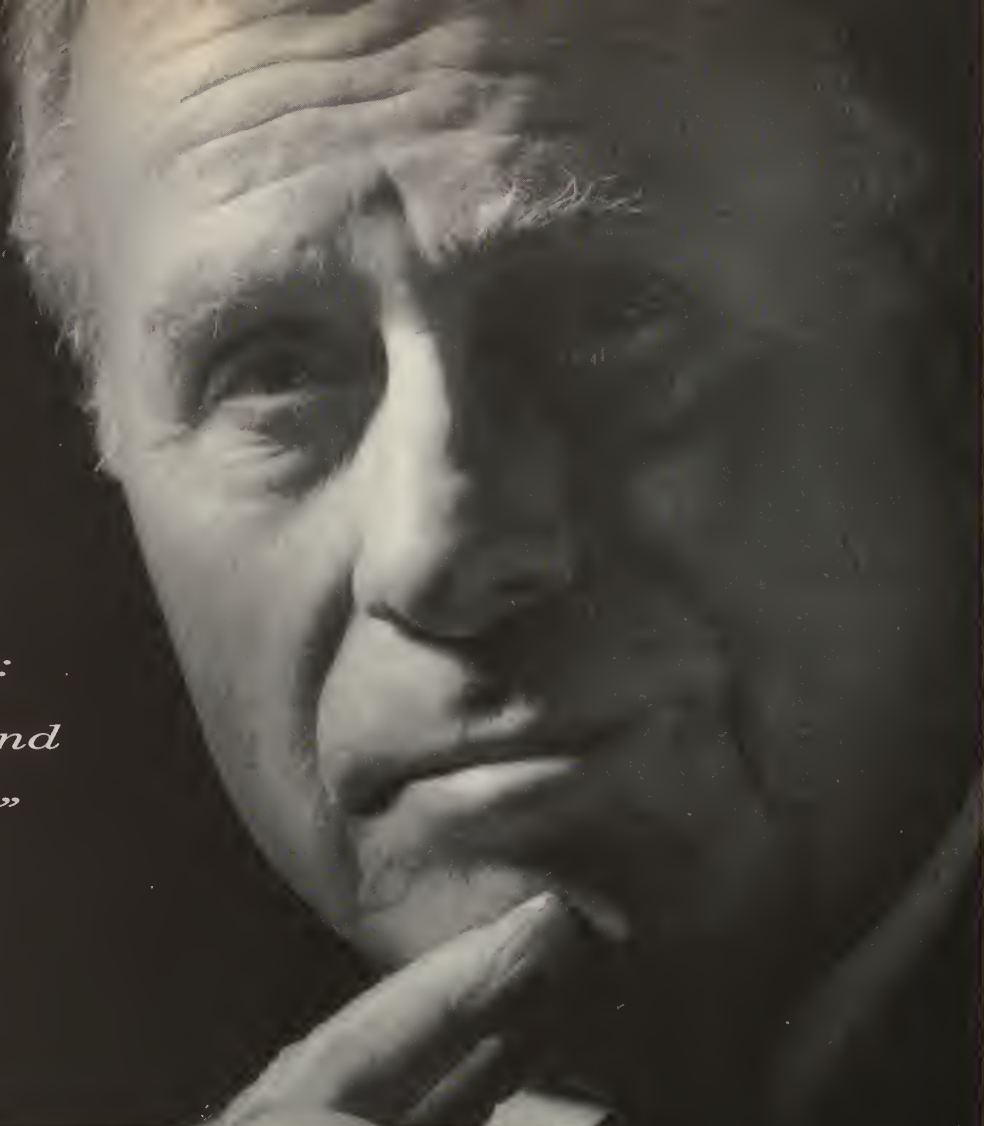
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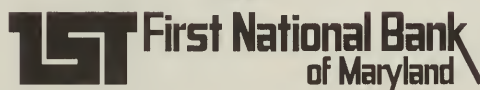


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


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Georgia Bond	★★★★	★★★★	—	—
Maryland Bond	★★★★	★★★★	★★★★	—
Maryland Short	★★★★★	★★★★★	—	—
New Jersey Bond	★★★	★★★★	★★★	—
New York Bond	★★★★	★★★★	★★★★	★★★
Summit Municipal Income	★★★★★	★★★★★	—	—
Summit Municipal Intermediate	★★★★★	★★★★★	—	—
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Rules and procedures take on an independent existence

Dr. Ronald J. Cohen's article, "Managed care and the physician-patient relationship: implications for peer review," (*Md Med J* 46;2:91-93) cut through the confused and fuzzy thinking so prevalent on this topic like a scythe through a field of ripe grain.

However, the bankruptcy of the present system was laid bare when he stated "...it becomes necessary to try to identify the licensed health care providers responsible for the formulation of the rules and procedures of the managed care organizations." I have on several occasions attempted to identify persons responsible for some of the more egregious "rules and procedures"

but have never been able to do so. Even if it were possible, using the authority of the BPQA [Board of Physician Quality Assurance] to identify such individuals, it seems to me they would more likely as not fall outside its purview, either geographically or professionally.

The end result of the situation is that the rules and procedures take on an independent existence subject to no control or oversight.

JACK C. CHILDERS, JR., M.D.
Baltimore, MD ■

As the practice of medicine changes, so must the laws regulating its practice change

Author responds

Ifully agree with Dr. Childers in his frustration at trying to identify those responsible for the way large health care organizations make decisions affecting how patients access care and how that care is delivered. However, as long as medical practice is licensed by the state, and as long as health care organizations require oversight by licensed physicians, the state maintains an interest in assuring that the medical care delivered by these health care organizations does not violate the prevailing standards of care as defined by the profession. At the time the current Health Occupations statutes were written, managed care was unknown to most legislators, and as the practice of medicine changes, so must the laws regulating its practice change. Otherwise, the state main-

tains the legal fiction of the traditional doctor-patient relationship, while increasingly, health care provider-patient interactions involve physician input in the board room and less in the consultation room. This does not mean that physicians should not participate in how organizations deliver care. They must, but those physicians who help to make organizational health care policy must also be held accountable for these decisions. The challenge is to decide how best to do this.

RONALD J. COHEN, M.D.
Chairman, Med Chi Peer Review Management Committee and assistant professor of neurological surgery, Johns Hopkins Medical Institutions ■

Physicians should expose capitation as the flawed system it is

My thanks to Drs. Seigel and Spiggle for their article, "Capitation: a moral issue in the managed care paradigm" (*Md Med J* 46;1:13).

I agree with all their points. As physicians we must never give up the role of patient advocate. It is clear that no one else will act on behalf of the patient. Insurance companies view patients as commodities; their singular goal is to cut costs with no remorse.

Physicians must learn to work toward a common goal together, that is, expose capitation for the flawed system it is. We have not been very good at working together in the past.

We need to:

1. Educate the public. It is my contention that if members of the public understood the thesis of capitation they would not want to be enrolled in such a plan. Organized medicine has failed to expose this concept for what it is. I doubt the HMO executives or members of Congress would want to be enrolled in capitation programs. Historically, I believe this is the first program that reimburses physicians for not rendering care—an unacceptable paradigm shift and an ethical dilemma for us.
2. Create a physician union. Maryland is small enough for us to achieve this. I would propose a union of physicians who refuse to participate in capitation programs on the grounds that quality of care may suffer. Enlightened businessmen have asked me how a group of intelligent, well-

educated people has allowed itself to be overrun by government agencies, insurance companies, and HMOs. The answer is because we are poor organizers. This needs to stop.

3. Lobby to reduce the size of medical school classes and post-graduate training programs. I believe that one of the main reasons that medical care has become costly and inefficient is because of the excess number of specialists. Computers and paraprofessionals make us all more efficient, and the need for more physicians is declining. Excess physicians have weakened our leverage with managed care companies. If you refuse to join, they can always find someone else who will. We are competing with ourselves for a finite number of slots and the businessmen know it.
4. Write to your federal and state representatives to encourage the use of tax deductible medical savings accounts. This program puts the decision making with the patient and the physician, and allows market forces to determine the value of a given service.
5. Never give up our role as patient advocate. If we relinquish that role, we might as well be washing cars.

If these suggestions sound too idealistic, then come up with better ones! It is time Maryland physicians started a dialog about the negative impact capitation has on our patients.

MICHAEL J. DODD, M.D.
Annapolis, MD ■

Capitation can be beneficial to providers and patients

The letter by Drs. Seigel and Spiggle in the January issue (*Md Med J* 46;1:13) is the typical approach by physicians who feel threatened by the emergence of capitation. True, there are inherent evils in capitation; but, potential for significant abuse exists in fee-for-service as well. Physicians, who are comfortable being independent and having a "blank check" with which to work, have a difficult time with the paradigm shift created by capitation, that is, the delivery of a product (patient care) for a set fee. Medicine is a business, and this concept is no different from any other in this regard.

The key to successful capitated contracts lies in the development of a partnership between the payer and provider, and in cases of specialty capitation, between the primary care provider and the specialist. A fair contract will provide guidelines to insure the quality of patient care. These guidelines will include parameters for specialty referral, surgical indications, and will clearly specify what services are included in the contract. Regular contract review to insure cooperation of all parties should be made, with specific penalties on the part of both provider and payer, should compliance not be met. A fair capitated rate, which will be fair to all parties and based on anticipated utilization, can then be determined. Any contract that is not a partnership, but is presented as

Editor's note: The much debated topic of capitation will be a primary focus at Med Chi's annual meeting, being held May 2-3, 1997. For more information call Med Chi at 410-539-0872 or 1-800-492-1056.

"here it is – take it or leave it," is not an offer to work together, but a threat, and should be avoided.

Maryland ENT IPA is a network of 65 independent otolaryngologists in Maryland, District of Columbia, and Northern Virginia organized for the very purpose of negotiation and administration of capitated contracts for otolaryngologic services. Our results with capitation using these principles have been mutually pleasing for both payer and provider and have evolved in to what we hope will be long term relationships. The true benefactor in these types of arrangements is the patient.

MICHAEL D. WEISS, M.D.
President, Maryland ENT IPA ■

Let's enter the third millennium a healthier nation by freeing ourselves from the scourge of tobacco

The American Cancer Society recently released the estimated cancer deaths for 1997. Almost one-third of these deaths are directly related to the use of tobacco. Isn't it time that we acknowledge that lung cancer has replaced breast cancer as the number one cancer killer of women? Isn't it time that we acknowledge that this fact has been true for *one decade* now? Isn't it time that we recognize that the 6 billion dollars spent on advertising by the tobacco industry plays a very major role in getting the populace to use tobacco products? Isn't it

time for the magazines geared to women to recognize the harm they are doing by carrying cigarette ads? Isn't it time for us to free ourselves from the control exerted by the tobacco drug lords via Madison Avenue and political contributions?

Isn't it time to think about entering the third millennium a healthier nation by freeing ourselves from the scourge of tobacco?

MIRIAM KLEBANER, M.D.
Smoke Free Maryland Coalition ■

Erratum

In "Spontaneous mediastinal hemorrhage: a case report with a review of the literature," published in the February 1997 issue of the *Maryland Medical Journal*, an incorrect computed tomography (CT) scan was printed for Figure 2. The following figure should have appeared:



Figure 2. CT of chest. There is posterior displacement of the trachea by a large anterior mediastinal density. The base of the heart is posteriorly displaced. The air-filled esophagus is seen behind the left main bronchus. A right pleural effusion is present.

The *Maryland Medical Journal* regrets the error.

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Toxic Exposure Advisory is a new monthly newsletter providing busy professionals with the latest information in toxicology. Topics will include the latest on both chronic and acute exposures; new medical monitoring technologies; emerging tests; current legal, medical, and regulatory issues; clinical and laboratory issues; proficiency testing issues; and pertinent environmental and medical information on toxicants. For more information, or to obtain a sample copy, contact Patrick Kilgariff, Journals Marketing Representative, Taylor & Francis, 1900 Frost Road, Ste. 101, Bristol, PA 19007-1598; telephone 215-785-5800, ext. 25.

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George Sopko, M.D., M.P.H., is among the authors of a study indicating that survival rates and quality of life are similar after five years whether a patient undergoes angioplasty or coronary bypass surgery. The study, which appears in the March 4, 1997, issue of *JAMA*, followed 1829 patients at 18 clinical centers for five years after the patients underwent either coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA). Dr. Sopko is from the National Heart, Lung and Blood Institute (NHLBI) in Bethesda.

Jorge D. Salazar, M.D., is among the authors of "Diagnosis of Injuries after Stab Wounds to the Back and Flank," published in the February issue of *The Journal of Trauma: Injury, Infection, and Critical Care*. This 10-year study evaluates the experience of selective management instead of mandatory exploration for patients with deep posterior wounds. Dr. Salazar is from the department of surgery at Johns Hopkins University.

Roger S. Blumenthal, M.D., Jeffrey A. Brinker, M.D., Jon R. Resar, M.D., Sean T. Gloth, M.D., Howard A. Zacur, M.D., Ph.D., and Gary Gerstenblith, M.D. are among the authors of, "Long-term estrogen therapy abolishes acute estrogen-induced coronary flow augmentation in postmenopausal women," published in the *American Heart Journal* (1997;133:323-328). Drs. Blumenthal, Brinker, Resar, Gloth, and Gerstenblith are from the division of cardiology, department of medicine, and Dr. Zacur is from the division of reproductive endocrinology, department of gynecology and obstetrics, Johns Hopkins University.

Robert W. Buchanan, M.D., Milton E. Strauss, Ph.D., Alan Breier, M.D., Brian Kirkpatrick, M.D., and William T. Carpenter, Jr., M.D. are the authors of "Attentional Impairments in Deficit and Nondeficit Forms of Schizophrenia," published in the March issue of the *American Journal of Psychiatry* (1997; 154:363-370). The study authors are from the Maryland Psychiatric Research Center on the grounds of Spring Grove Hospital, Baltimore.

Roger S. Blumenthal, M.D., is the lead author in a study showing that long-term estrogen replacement therapy after menopause may reduce heart risk not only by lowering blood-fat levels, but also by increasing blood flow to the heart and causing the blood vessels to stay open wider and longer. The study appears in the March issue of *American Heart Journal*. Dr. Blumenthal is an assistant professor of medicine at Johns Hopkins Medical Institutions.

Jessica L. Bienstock, M.D., is lead author of a study indicating that young physicians who undergo a rigorous formal training program in ultrasound testing on pregnant women are better skilled at this procedure than young physicians without such training. Results of the study, presented at the annual meeting of the Association of Professors in Obstetrics and Gynecology and Council on Resident Education in Obstetrics and Gynecology, show that residents who underwent a formal training program in obstetrical ultrasound had a mean score of 67% on a practical exam, compared with 53% by obstetrics residents who did not. Dr. Bienstock is an instructor in gynecology/obstetrics at the Johns Hopkins Medical Institutions.

If you have been recently published and would like to be listed in Maryland Medicine in Review, send a photocopy of your published article to:
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Enhancing patient outcomes through an understanding of intercultural medicine: Guidelines for the practitioner

Carol Jack Scott, M.D., MEd, FACEP

Dr. Scott is a clinical assistant professor in the division of emergency medicine, department of surgery, University of Maryland Medical Center. She is also president of The Medical Education Group in Baltimore.

ABSTRACT: *As cultural and ethnic diversity increase within American society, physicians face new challenges in recognizing patients' culturally defined expectations about medical care and the cultural/ethnic dictates that influence physician-patient interactions. Patients present to practitioners with many mores related to concepts of disease and illness, intergenerational communication, decision-making authority, and gender roles. In addition, many cultural groups follow folk medicine traditions, and an increasing number of Americans seek treatment by practitioners of alternative therapies before seeking traditional western medical attention. To facilitate patient assessments, enhance compliance with health care instructions, and thus achieve the best possible medical outcomes and levels of satisfaction, practitioners must acknowledge and respect the cultural differences patients bring to medical care environments.*

Physicians diagnose and treat disease (i.e., abnormalities in structure and function of body and system), but patients have symptoms and suffer illness (i.e., changes in body states). While physicians are thinking in terms of anatomy and physiology, their patients may be thinking in terms of yin and yang, thick or thin blood, or hot and cold.

Health care outcomes, clinical and non-clinical, can be improved by considering patients' cultural beliefs about health and illness.^{1,2} To enhance clinicians' awareness and appreciation of cultural diversity, this article presents guidelines that can and should be applied to all ethnic and culturally distinct groups (but that are not intended to describe specific cultural perspectives or practices). General principles of cultural compe-

Table 1. Sizes of racial groups in Maryland & Baltimore City (1990 U.S. Census data and nomenclature)

	Maryland	Baltimore City
• White	3 393 964	287 753
• Black	1 189 899	435 768
• Asian/Pacific Islander	139 719	7 942
• American Indian/Eskimo/Aleut	12 972	2 555
• Other	44 914	1 996
Total	4 781 468	736 014

The numbers of people of Hispanic background totaled 125 102 in Maryland and 7 602 in Baltimore City.

tence will be presented and their adoption in medical practice encouraged.^{3,4}

Demographic changes

The demographic characteristics of the United States are changing rapidly. The Bureau of the Census projects that, by the year 2000, approximately 25% of the American population will be members of current "minority" groups. The diversity of Maryland and Baltimore City is portrayed numerically in **Table 1**. Providers must be aware of the cultural diversity within the communities they serve and must enhance their awareness of the various cultural groups' unique characteristics that affect communication and health care outcomes.

Culture and ethnicity: definitions

Culture encompasses the concepts, beliefs, and values that members of a social group use to give order and thus meaning to their relationships and the physical world. The concept of culture includes the customary beliefs, social norms, and material traits of a racial, religious, or social group.⁵ Incorporating the concept of cultural identity, Brislin⁶ describes culture as the widely shared ideals, values, formation and use of categories, assumptions about life, and goal-directed activities that become unconsciously or subconsciously accepted as "right" and "correct" by people who identify themselves as members of a society.

Ethnicity is a sociological construct, highly correlated with behavioral and cultural phenomena. Schermerhorn⁷ defines an ethnic group as a collectivity within a larger society, having real or assumed common ancestry, memories of a shared past, and a cultural focus on symbolic elements such as kinship patterns, physical contiguity, reli-

gious affiliation, language or dialect forms, tribal affiliation, or phenotypic features.

The terms *race* and *ethnicity* are often used interchangeably, but they are far from synonymous.⁸ The concept of race comes from taxonomic concepts applied in botany and zoology. It is defined as "any of the major biological divisions of mankind, distinguished by color or texture of hair, color of skin and eyes, stature, bodily proportions, etc."⁹ The three traditional categories of race (caucasoid, negroid, and mongoloid) imply genetic homogeneity among persons grouped according to visible characteristics. The "tidiness" of these distinctions does not convey the wide range of practices and beliefs within them. For example, a Nigerian newly immigrated to the United States, a Haitian, and an African-American are "racially identical" (i.e., black), but they do not share biologic inheritance, nutritional habits, or beliefs about medical care. Racial lines are cultural rather than scientific constructs. The terms "black" and "white" tell more about how American society has been structured than about medically relevant realities. In fact, the mention of race in oral and written medical presentations has been discouraged.^{9,10}

In contrast, ethnicity often does influence general styles of interaction, attitudes toward authority figures, sex-role allocations, and ways of expressing emotion and asking for help, which carry over into health care situations. For example, Blackhall and colleagues¹¹ studied elderly patients' preferences about diagnostic disclosure and end-of-life decision making. They found that Korean-Americans and Mexican-Americans were more likely to desire a family-centered model of medical decision making and that African-Americans and European-Americans favored greater patient autonomy. The authors suggest that heightened awareness of such ethnic differences will lead physicians to ask patients if they wish to receive information themselves and make their own decisions or if they prefer the families assume those responsibilities.

In summary, patients' culture and ethnicity provide their conceptual framework for encounters with the health care system.

Concepts of disease and illness

Western medical thought defines "disease" as a malfunctioning of biologic and physiologic processes. The term "illness" represents personal, interpersonal, and cultural responses to disease and discomfort; it is used according to culturally acquired perceptions and attitudes, as well as complex family and social structures. Concepts of disease

and illness intertwine to create the multi-levelled concept of "sickness."

The classification of symptoms into illness categories is also ethnically/culturally bound. Examples of culture-specific "syndromes" are *susto* among some Mexican-Americans, *evil eye* among various circum-Mediterranean groups, *Arctic hysteria* among Eskimos, and *battement de coeur* among Haitian-Americans. In addition, ethnicity correlates with conceptions of the causes of disease and illness, for example, the idea of balancing opposing forces such as Chinese yin and yang, East Indian Ayurveda and other humoral systems, hot/cold theories among Hispanics, beliefs about God's punishment or possession by the devil among some southern blacks, and gas and blood perturbations among Haitian-Americans.

A disease and illness model

A four-stage model of disease and illness can be applied by physicians during evaluation of patients from most cultures. Physicians and patients may have different health belief models. Physicians tend to focus on recognition of disease, and the health care system tends to disregard illness as a legitimate clinical concern. When only disease is addressed therapeutically, patients and their families tend to be less satisfied with medical care received. Discrepancies in health belief models translate into different views of clinical reality, which can lead to poor patient compliance with medical advice and to lawsuits. Despite the applicability of each of the following four steps in the clinical setting, each carries the potential for cross-cultural misunderstandings or clashes:

Onset. Onset is the time when a person experiences the first symptoms or becomes aware of a problem. The concepts governing awareness are culturally dictated. If the onset is slow and insidious, the person may not be conscious of symptoms or will wait with the hope that the disability or discomfort will go away. If the onset is acute, the person recognizes more immediately that illness has occurred and help must be sought. This stage, and its inherent variability in length, is the prelude to legitimization of illness.

Diagnosis. In the diagnostic stage of the illness experience, an effort is made to identify the disease. With diagnosis, the illness is culturally recognized and the person's role is sanctioned. On the other hand, a diagnostic workup is an unfamiliar (and difficult) experience for most people: patients are expected to describe very personal matters to strangers and are often subject to painful and frightening procedures.

Table 2. Constructing clinical reality: Understanding the patient's concept of disease and illness

- ♦ What do you think has caused you to be sick?
- ♦ Why do you think your sickness started when it did?
- ♦ What do you think your sickness does to you?
How does it work?
- ♦ How sick are you?
- ♦ Do you think you are going to be sick for a long time?
Or do you think you will get better soon?
- ♦ Will you get better on your own?
- ♦ Has anyone else you know ever had the same problem?
- ♦ What kind of treatment do you think you need?
- ♦ What are the most important results you hope to get from treatment?
- ♦ What are the major problems this illness has caused you—at home and other places?
- ♦ What do you fear most about what is going on?

Patient status. A person who becomes a patient must not only give into the demands of his or her physical condition but also, in many cases, become dependent on others for the basic needs of daily life. The patient must also adjust to the social aspects of being ill, accepting the culturally defined role. Loss of normal control poses a variety of difficulties, especially in the emergency care setting, where the patient's environment is highly structured and boundaries are determined by health care providers, not by the patient.

Recovery. In the final stage, recovery, patient status is relinquished and the person resumes pre-illness roles and activities. If recovery cannot be complete, conflict can emerge as the person copes with an undesirable change in body image or functional ability. From the medical care provider's viewpoint, the patient has recovered, because his or her body no longer has the symptoms of the acute illness that prompted the person to seek help. From the former patient's perspective, illness persists because of immediate and long-term disability. As stated before, the clinician must distinguish illness from disease but should not regard the two as distinct entities.

Sources of treatment

Individuals seek intervention for the illness/disease process in any of three domains: popular (e.g., self-treatment, family care, self-help groups), folk (e.g., religious practitioners, healers), and professional (e.g., western-trained physicians, physicians' assistants, nurses). Most illness episodes never reach the professional domain: 70% of patients use popular resources before seeking professional health care services.¹²

Table 3. General principles of knowledge/attitudes/skills needed for cultural competence

Intercultural knowledge

- ♦ American society is heterogeneous; physicians and patients are often of different cultural backgrounds.
- ♦ Culture is important in the identity of all patients as well as all physicians.
- ♦ The physician's own culture and the culture of medicine have important impact on his or her relationship with patients.
- ♦ Communication of cultural understanding and respect is essential for establishing rapport and confidence.
- ♦ Culture-related concepts (e.g., explanatory model of illness) and behaviors (such as religion, diet) affect patients' acceptance of and compliance with prescribed therapy.
- ♦ Nonverbal and verbal communication may differ from culture to culture.
- ♦ Each individual patient is a unique combination of cultural, family, social group, and idiosyncratic beliefs.
- ♦ A physician should communicate with a patient in a language in which the patient is fluent.
- ♦ Trained medical interpreters have an important role in the bilingual medical encounter; such personnel, instead of family members, should be asked to facilitate communication whenever possible.
- ♦ Culture-related stresses and tensions can induce illness.
- ♦ Differences in the clinical epidemiology of common illnesses may be related to culture.

Intercultural attitudes

- ♦ Recognition of the importance of a patient's cultural background and environment in constructing an approach to an illness
- ♦ Acknowledgment of patient's role as an active participant in his or her own care and as an "expert consultant" in conveying cultural information
- ♦ Work to overcome language and cultural barriers in providing care; provide support and advocacy for patients.

Intercultural skills

- ♦ Communicate an interest in and respect for the patient's culture.
- ♦ Tactfully and respectfully elicit general cultural information.
- ♦ Elicit patient's understanding of and beliefs about illness or health problems.
- ♦ Elicit and consider information regarding possible culture-related health problems.
- ♦ Interpret verbal and nonverbal behaviors in a culturally relevant manner.
- ♦ Negotiate a culturally appropriate health care plan with patient and family as partners.
- ♦ Demonstrate the ability to work as a team with a medical interpreter in the bilingual medical encounter.

Categories of cultural information needed for particular cultural groups encountered frequently

- ♦ Predominant cultural values
- ♦ Traditional health practices and health beliefs
- ♦ Family structure: patriarchal or matriarchal; nuclear or extended; roles of individual members
- ♦ Community structure and the roles of community leaders
- ♦ Religious beliefs and their effect on health care beliefs and practices
- ♦ Customs and attitudes surrounding death
- ♦ Nature and significance of common verbal and nonverbal communication styles
- ♦ Common dietary habits and nutritional content of foods
- ♦ Awareness of "culture shock," particularly in relation to patients entering modern health centers
- ♦ Awareness of prevailing intercultural tensions and psychosocial issues

Modified from Goldstein E, Bobo L, Womeodo R, Kaufmann L, Nathan M, Palmer D, Scott CJ. *Intercultural medicine*. In Jensen NM, Van Kirk JA (eds). *A Curriculum for Internal Medicine Residency*. Philadelphia, American College of Physicians, 1996.

During an illness episode, an individual may use various sources of care. A sick person may seek treatment from a folk therapist to eliminate the cause of the disease while simultaneously receiving treatment from a biomedical practitioner for relief of symptoms.

Although it is uncommon for folk remedies to have adverse effects, some treatments may be hazardous—for example, geophagia (the ingestion of earth or clay), a folk practice in Africa and the American South. Other folk practices produce skin lesions that can be mistaken for signs of abuse. Examples are the Southeast Asian practice of "coining" (*cao gio*), the Chinese practice of moxibustion, and the Mexican-American practice of cupping.

Folk illnesses may be cultural interpretations of pathophysiology that may, in fact, need medical attention. For example, the Mexican folk illness *casida de mollera* (fallen fontanelle) identifies significant dehydration in infants.

Concepts within folk medicine practices may hold important clues for patient assessments conducted by physicians.¹³ Familiarity with these concepts can open communication pathways leading to improved patient care and satisfaction with the care received.

Increasing numbers of people are being treated by specialists in alternative/complementary therapies (e.g., massage, acupuncture). A 1994 study of 3,789 adult

Americans, sponsored by the Commonwealth Fund,¹⁴ revealed the following patterns: Minority groups reported the use of alternative medicines twice as often as whites (25% versus 14%). Herbal medicine had been used during the year preceding the survey by about one third of participants of Chinese and Korean heritage and by 12% of whites. Twenty-two percent of Koreans used acupuncture, compared with 1% of all groups. And whites were twice as likely to have seen a chiropractor in the past year than were minority adults (13% and 7%, respectively).

Each of the three interventional domains has its unique explanatory system, social roles, interaction settings, and institutions. Clearly, when a patient comes to a traditional professional setting, a *cultural construct of clinical reality* must be negotiated between the physician and the patient.¹⁵

A dialogue between the patient and provider, which is needed to construct a clinical reality, can be facilitated by the questions ("probes") listed in **Table 2**. Gaining facility with this interaction method is a core element of the competencies needed to effectively provide care for diverse populations. Further, and more importantly, this interaction method supports patients' empowerment to better care for themselves.¹⁶

It is beyond the scope of this article to describe all of the competencies needed for cross-cultural medicine; however, general principles of cultural competence are listed in **Table 3**.

Use of translators

When caring for patients for whom English is not the primary language, several issues emerge. First, providers must realize the interaction is always *bicultural* and not merely *bilingual*. Successful communication requires more than mechanical translation.¹⁷ Second, the physician should not assume that a bilingual person wants to speak in his or her native language. Patients often can understand more English than they are able to express. Third, if an interpreter is necessary, a professional is preferred or, at minimum, a person with a biomedical background. If only nonprofessional employees are available in a practice setting, brief training to enhance specific skills within this "pool" is recommended. The clinician should use clear, simple English and should establish rapport and mutual respect with the translator. Debriefing with the translator after a session is critical to foster understanding of potential intercultural misinterpretations.

Table 4. Practical guidelines for a culturally appropriate approach to health care

- ♦ Recognize intraethnic variation.
- ♦ Recognize ethnic- and culture-bound gender role norms.
- ♦ Elicit and understand the patient's concept of the sick episode.
- ♦ Identify sources of discrepancy between physician's and patient's concept of disease and illness.
- ♦ Validate the patient's perspective.
- ♦ Provide education and work within the patient's conceptual system.
- ♦ Negotiate a "clinical reality" on which patient and physician can base an approach to treatment.
- ♦ Validate resolution of the patient's concerns about illness and disease at the end of the encounter.
- ♦ When the assistance of a translator is required, encourage use of the patient's own words.
- ♦ Employees who will serve regularly as translators, but who are not trained in biomedicine, should complete a brief program in cultural sensitivity/competence.
- ♦ Provide patients with cards printed with routine requests in English and their native language.
- ♦ Consider ethnically and culturally acceptable diets, food preferences, and religious beliefs.

Strategies for patient care

Certain strategies for patient-physician interaction are applicable to all ethnic and cultural groups (**Table 4**). These guidelines describe an approach that can be individualized for each patient and thus enhance information gathering, compliance with medical advice, and patient satisfaction with the health care experience.

During history taking, the physician should elicit information about the patient's concept of disease and illness. Although there are many communication issues in the managed care environment,¹⁸ particular care must be taken to communicate nonjudgmental attitudes as well as respect for, and genuine interest in, the patient. Time spent listening will enable the provider to learn how to help the patient and will establish trust, a key factor in achieving patient satisfaction.

When discrepancies emerge between the physician's and the patient's concepts of disease, clear communication is essential. The patient and physician must be able to understand the terms being used by each other. A patient who might be described as "noncompliant" actually may be "following the rules," but rules based on a different set of principles.¹⁹ Compliance with the prescribed treatment plan is much more likely if the physician is aware of the patient's health beliefs.

Although concepts of disease vary across ethnic groups, some general ideas are somewhat consistent. The physician should give special consideration to these themes/concepts

while talking with—and listening to—patients and engaging them in treatment plans. Clinicians should also be open to intraethnic variation: A common assumption is that ethnic groups are monolithic and uniform in beliefs and behavioral norms. Such assumptions ignore within-group diversity and lead to simplistic ideas and even stereotyping.

Central role of family. In many ethnic groups, medical decision making, receiving and disclosing news, and coordinating care are concerns and responsibilities of the group, not the individual. Related cultural values are the social role of women, attitudes toward sexuality, and intergenerational relations.

Mental health. Cultural patterns for expression of emotions and the value placed on “talk therapy” should be considered. Some patients (particularly immigrants) may present with long-term mental health effects resulting from events such as natural disasters, political upheaval, or war. Presentations may include depression, apathy, and somatization.

Medical pluralism. Alternative healing systems may alleviate physical symptoms, and they can address psychosocial issues ignored by biomedicine. Western medicine, alternative/complementary therapy, and folk medicine should not be considered mutually exclusive.

Prevention. Western prevention strategies must fit with cultural beliefs. Cultures with health belief systems that balance opposing forces (e.g., Chinese yin and yang) naturally provide behavioral principles and thus means of preventing excesses.

Finally, five therapeutic issues recur across many ethnic groups. Being aware of them and exploring culturally based expectations with patients will enhance clinicians’ understanding when negotiating strategies for achievement of outcomes:

- ▶ Relief of pain or other symptoms
- ▶ Anxiety-provoking symptoms
- ▶ Fear of treatment
- ▶ Interference with role responsibilities
- ▶ Interference with valued activities

In summary, culturally and ethnically bound beliefs and behaviors apply strong influences on patient-physician interactions and their outcomes. Physicians bear the responsibility for fostering effective communication with patients. Patient care can be managed more effectively, with the patient and practitioner as partners, if providers bring heightened awareness of the culture and experience of the patient to assessment and treatment environments.^{20,21}

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THE USE OF ANTI-AGING HORMONES

*Melatonin, growth hormone, testosterone,
and dehydroepiandrosterone: Consumer
enthusiasm for unproven therapies*

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ABSTRACT

Since ancient times, humans have been concerned with developing and preserving youthful vigor. Today, there is enough understanding of the aging process to attempt to delay it. This review considers four popular and easily obtainable anti-aging hormones: melatonin, growth hormone, testosterone, and dehydroepiandrosterone (DHEA). Many of the benefits of using these hormones, which are promoted in the lay literature, are based on animal studies and weak associations. This review critically examines the scientific literature. At this time, there is insufficient evidence to recommend these hormones as therapies for aging, and there are potential risks from their use. The information provided here will help physicians discuss the use of these hormones with inquiring patients.

Introduction

In 1869, a young physiologist, Charles-Edouard Brown-Sequard, transplanted young guinea pig testes into a dozen old dogs. Twenty years later, feeling that he was failing in physical and mental vigor, he gave himself subcutaneous

injections of an extract of crushed dog and pig testicles. He reportedly regained much of his former strength. From that moment until his death, he was obsessed with rejuvenation. Because of Brown-Sequard's tremendous reputation, the rest of the medical world was very excited about his findings. More than 12 000 physicians began administering the extract to patients for everything from cancer to tuberculosis.¹

Today, fascination with anti-aging remedies has reached tremendous levels. Americans spend about \$6 billion annually on health supplements, and the market is growing 20% every year. In 1994, when Congress passed the Dietary Supplement Health and Education Act (DSHEA), sponsored by Senators Orrin Hatch and Ted Kennedy, vitamins, minerals, and herbs became food supplements, not drugs, thus reducing Food and Drug Administration (FDA) control over them.²

The biology of aging

Theories to explain the process of aging abound. There are, however, a few fundamental observations. First, humans and animals share a common loss of physiological reserve and share an exponentially increasing vulnerability to life-threatening illnesses. These processes include kidney degeneration, susceptibility to neoplasia, neurological deficits, weakening of immune function, decline in cardiovascular capabilities, alterations in collagen and crystalline structure, and changes in protein synthesis and degeneration rates. Second, the rate of aging, which varies between species, is under genetic control. The maximum life-span potential goes from 3 to 5 years in rodents to 115 years in humans. And third, caloric restriction retards most aspects of aging in mammals. In rodents, it was found that caloric restriction to 60% of the *ad libitum* intake (as long as micronutrients are provided to prevent malnutrition) will increase the maximum life span as much as 40%.³

Several models have been proposed to explain these observations. Orgel's "error catastrophe" model implicates errors in DNA transcription and RNA translation causing structural alterations in proteins. Many theories involve a "master clock" mechanism, in which damage to some organ, cell-type, or molecule helps potentiate age-related changes. The human circadian clock is thought to be located in the suprachiasmatic nucleus of the hypothalamus. The "free radical" theory suggests that highly reactive by-products of oxidative metabolism react with proteins, DNA, and lipids to generate dysfunctional molecules that interfere with cells.³

Melatonin

Melatonin is synthesized from tryptophan, mostly by the pineal gland, which secretes it at night. Beta-adrenergic

blocking agents or light cause a sharp decline in secretion. When secreted, luteinizing hormone and growth hormone levels are decreased. It induces sleepiness and increases the number of alpha waves on the EEG. Some report that it produces a sensation of well-being and elation.⁴

Melatonin is marketed as a dietary supplement under the DSHEA and is not reviewed by the FDA for efficacy or safety. Manufacturers are not required to present evidence of purity or even prove how much, if any, melatonin is in their product. Most marketed melatonin is synthetic, but some is extracted from bovine pineal glands. Concerns about the purity of unregulated products have led to reports of independent laboratory consultants chemically analyzing some products and finding uncharacterizable impurities.⁵ The potential for an epidemic like eosinophilia-myalgia syndrome (EMS) may result from such impurities. Since October 1989, EMS has killed 40 Americans and has left about 2 000 others with moderate to severe symptoms. EMS resulted from a toxic substance produced during the manufacture of L-tryptophan by a Japanese firm. The FDA had previously considered L-tryptophan a generally safe food ingredient for use as a dietary supplement.⁶

There is a growing amount of literature on the physiology of melatonin. At least two melatonin receptor subtypes have been identified and cloned in several species. Melatonin is lipophilic and potentially interacts with a number of intracellular targets. It acts as a calcium-calmodulin antagonist, which indicates a potentially important role in cellular homeostasis. It acts as a potent (perhaps the most potent known) scavenger of hydroxyl free radicals and can accumulate within the nucleus of cells, where it can bind nuclear proteins and DNA, perhaps functioning in a protective role. Melatonin receptors have been identified in the suprachiasmatic nucleus of rodents (not yet humans). Thus aging, which is associated with a reduction in output from the circadian clock, may be mediated by melatonin.⁷

Walter Pierpaoli of the Italian National Research Centers on Aging, author of *The Melatonin Miracle*, believes that aging is undeniably initiated in the pineal gland. He cites a study showing that exogenous melatonin increases the life span of aged mice by 25%. Pierpaoli believes correcting secondary deficiencies (i.e., replacing DHEA and other hormones) which are a consequence of aging is valid adjuvant therapy, but only modifying central pineal function can

significantly control aging.⁸ Several studies do show a decline in nocturnal melatonin levels in aging.^{9,10}

Melatonin also interacts with the immune system. Pinealectomy causes decreased reactivity of immune markers in laboratory animals. These effects are reversed with administration of melatonin. Melatonin saved mice from death by the encephalomyocarditis virus that killed most of the controls. Several human trials of melatonin in cancer patients have been reported. Controls were not used in the studies, and no significant conclusions were reached.⁹ Underfeeding laboratory animals, as mentioned previously, increases longevity. Studies have shown that underfed rats have higher melatonin levels. There is a report of melatonin inhibiting platelet aggregation in humans, suggesting a theoretical beneficial effect on coronary artery disease.¹¹ In another small study, impaired secretion of melatonin is associated with coronary artery disease.¹² These effects, if confirmed, may be due to the suppression of catecholamines by melatonin.

Possible adverse effects of taking melatonin include contamination by a toxic impurity, resetting the body's circadian clock incorrectly, and daytime drowsiness.¹³ An MIT research team demonstrated a dose of 0.1 to 0.3 mg provides maximum benefit; this is less than the over-the-counter dose. Mental impairment and severe headaches are reported at these higher doses.¹⁴

The melatonin market is valued at \$25 to \$30 million.¹⁵ United States sales now exceed those of vitamin C.¹⁶ As companies escalate production to meet demand, safety issues become more concerning. Other countries have restricted access to melatonin. In England, melatonin is available only by prescription. Health Canada has banned the sale and production of the hormone, driving consumers to look for illegally stocked supplies or go across the border to the United States.¹⁷ For those who do not want to consume a non-FDA-approved substance, endogenous melatonin levels can be naturally boosted by increasing daytime exposure to sunlight, avoiding bright lights in the evening, quitting smoking, minimizing alcohol consumption, and avoiding night shift work or crossing time zones.¹⁷

In conclusion, several small placebo-controlled trials demonstrate that melatonin has the potential to help people with sleep disorders and jet lag.⁵ As listed above, there are many studies showing anti-aging effects of melatonin in laboratory animals and *in vitro* models. There is no proof, however, that melatonin will prolong life, boost the immune system, prevent cancer, or reverse aging in humans. There are serious questions about the safety of unregulated melatonin products sold in the United States.

Dehydroepiandrosterone

Claims about melatonin such as increasing vigor, boosting the immune system, fighting cancer, and slowing aging are also made for another fat hormone, dehydroepiandrosterone (DHEA). DHEA, called the mother steroid because of its abundance in the body and its use as a precursor for other steroids, has unclear physiological importance. It has both estrogenic and androgenic effects but has no known target organs or receptors.¹⁸ There is no evidence for a vital function for DHEA; patients without functioning adrenals survive without its replacement. DHEA is a neuroactive neurosteroid in the rat, inhibiting and potentiating a number of central nervous system receptors, but effects in the human brain are unclear.¹⁹

DHEA's role in aging stems from the observation that in humans its levels dramatically decline with age.²⁰ Morales and colleagues²¹ conducted a 6-month placebo-controlled trial and found a remarkable increase in perceived physical and psychological well being (i.e., improved quality of sleep, greater energy, and increased ability to handle stress) in 67% of the men and 84% of the women. As for objective measurements, insulin-like growth factor-1 (IGF-1) levels increased in the study patients. In another study, lean body mass and muscle strength increased and fat body mass decreased.²² Perhaps the psychological changes were mediated by direct neurotrophic effects. The elevation of IGF-1 levels may account for DHEA's potential effects on muscle mass, immune function, and other physical parameters.

Data on the effects of DHEA on the immune system are mostly derived from animal studies that show beneficial modulation of immune function. Rodents have almost unmeasurable levels of endogenous DHEA, so results in these studies are not directly transferable to humans. Three small human studies suggest possible beneficial effects on the immune system.^{22,23}

It has been proposed that DHEA could be protective against certain cancers either by immune mechanisms or by anti-proliferative effects. Again, anti-cancer effects have been shown only in animal studies. In one case-control human study of 15 women with premenopausal breast cancer, the mean serum level of DHEA was 10% lower among women with breast cancer, but the results were not statistically significant.²⁴

Declining DHEA levels have also been implicated in the age-related increase in cardiovascular disease. A prospective study of about 2 000 men and women initially suggested that high DHEA levels were protective against coronary artery disease. This finding generated much publicity, but

final analysis of the full cohort adjusted for other risk factors failed to show statistically significant differences.²⁵ A three-week study of the effects of 50 mg of DHEA given to 11 postmenopausal women showed an enhancement in insulin sensitivity and a decrease in triglyceride levels.²⁶ In fact, some investigators believe the beneficial effects of DHEA are mediated by insulin levels.²⁷ Perhaps DHEA plays a role in lipid or glucose metabolism, but a significant effect on coronary disease has not been shown.

In the 1970s and 1980s, DHEA was a popular weight-loss aid available at health food stores. In 1985, the FDA banned over-the-counter sales due to lack of evidence for efficacy in weight loss and lack of data on safety.²⁸ Some manufacturers marketed Mexican yam extracts during this time, claiming they contained precursors to DHEA. Scientists, however, believed little of the extracts were converted to DHEA in the body. Currently, the FDA considers DHEA a "dietary supplement" as long as therapeutic claims are not made concerning its use.²⁹ A 25 mg bottle of 30 tablets ranges in price between \$7 and \$15. Unlike melatonin, supplies are abundant.

There is not enough evidence to recommend routine treatment with DHEA. Many consider the evidence cited here as less than objective, because many of the studies were published at a New York Academy of Sciences conference, where about one quarter of the participants were taking DHEA.³⁰ In summary, DHEA levels decrease with age, many studies show potential benefits to supplementation, and replacement therapy carries little risk. Anecdotal side effects include hirsutism, acne, hair loss, deepening of voice in women, and a report of hepatitis. Theoretically, the potential exists for the stimulation of hormonal responsive tumors such as breast and prostate cancer.³¹ If a patient requests DHEA therapy, an untested rationale may be to check the DHEA level, give replacement only if the level is low, and do not give replacement unless a serum prostate specific antigen is normal and breast cancer risk is low.³²

Testosterone

Estrogen hormonal replacement in aging women has become common practice. Women have a rapid decline in estrogen levels at menopause; no discrete event marks a transition to male senescence. Male sexual function does, however, decrease with age. The rigidity of penile erection and force of expulsion of ejaculate are greatest and the refractory period is shortest at age 17. Leydig cell function and sperm production decrease. There is also a significant increase in basal luteinizing hormone levels and a diminished testosterone response to exogenous gonadotropin. A

loss in the testosterone circadian rhythm also occurs in older men. However, most studies show that older men have testosterone levels similar to younger men.³³ But the levels of sex hormone binding globulin increase, thus the amount of the free biologically-active testosterone is lower.³⁴

Androgens have many biological effects. They are required for the maintenance of the male reproductive organs. Testosterone replacement in hypogonadal males increases interest in sexual activity. Testosterone increases the size of muscle cells. Dihydrotestosterone stimulates sebum production and hair growth. Androgens increase the synthesis of clotting factors and erythropoietin, decrease the levels of high density lipoprotein, increase the levels of low density lipoprotein, and stimulate the proliferation of bone cells. They also are associated with changes in immune function because testosterone improves the symptoms of autoimmune disease in Klinefelter's syndrome. Similarly, women have greater cell-mediated and humoral immunity than men with a greater incidence of autoimmune disease.³³ Hence, the diverse effects of androgens may impact on aging through various mechanisms.

Current clinical indications for testosterone therapy are treatment of men with documented testosterone deficiency. Most men notice increased libido, energy, and strength within days or weeks of starting therapy. Side effects include acne, fluid retention, gynecomastia (from peripheral aromatization to estrogen), sleep apnea, and erythrocytosis. Peliosis hepatis, or hemorrhagic liver cysts, can occur. Complications of supraphysiologic doses include decreased testicular size, azoospermia, cholestasis, jaundice, hepatic failure, dyslipidemia, coronary artery disease, increased prostate specific antigen, and increased aggression.³³

The study showing supraphysiologic doses of testosterone increase muscle size and strength was only recently published.³⁵ Giving 600 mg of testosterone or placebo for 10 weeks to a group of men randomized to exercise or no exercise led to increases in fat-free mass. The combined regimen of testosterone and exercise resulted in an increase of 6.1 kg of fat-free mass; testosterone alone led to a 3.2 kg increase; and exercise alone led to a 2.0 kg increase. Exogenous androgens have been advocated for use in postmenopausal women as part of hormonal replacement therapy and have been found to improve sexual functioning, increase energy levels, and alleviate depression and headaches.³⁶

In summary, testosterone demonstrates a benefit on muscle mass, but its use as an anti-aging hormone is limited by its unpleasant side-effects, unfavorable lipid effects, and its increasing prostate cancer risk.

Growth Hormone

Common themes in the above discussions of melatonin, DHEA, and testosterone also resound in the discussion of human growth hormone, which is known to stimulate skeletal and muscle growth and regulate carbohydrate and fat metabolism. These effects are mediated by growth-promoting peptides called somatomedins; the best known is somatomedin-C or insulin-like growth factor-1 (IGF-1).

Lean body mass in men over 50 years old decreases by 0.6% each year with reciprocal expansion of adipose mass. The shrinkage of lean body mass reflects atrophy of skeletal muscle, skin, and visceral organs.³⁷ Interest in using growth hormone to reverse these changes of aging was sparked by a study of 12 healthy men, 61 to 81 years old, who received subcutaneous injections of growth hormone for 6 months. Mean plasma IGF-1 levels rose to the youthful range. There was an 8.8% increase in lean body mass, a 14.4% decrease in adipose tissue mass, a 1.6% increase in average lumbar vertebral bone density, and a 7.1% increase in skin thickness. The cost of the administration would be about \$15 000 a year.³⁸

Theoretical side effects (as observed in patients with acromegaly) include impairment in carbohydrate metabolism (i.e., hyperinsulinemia, glucose intolerance, and diabetes), adverse effects on the musculo-skeletal system (i.e., arthritis and arthralgias), and adverse effects on the cardiovascular system (i.e., hypertension, edema, and congestive heart failure).³⁹ Patients with acromegaly also have an increased risk for certain cancers.⁴⁰ One study confirmed the effects of growth hormone on lean body and adipose tissue mass. The frequency of side effects in one group taking a rather low dose for 4 months was quite high: carpal tunnel syndrome (25%), fluid retention/edema (15%), and arthralgias (15%).⁴¹

Children who are growth hormone deficient receive replacement therapy until they reach a reasonable adult height, because growth hormone is absolutely required for linear growth. This replacement therapy is not continued in adults. However, recent data show that these adults have abnormalities in body composition that correct with growth hormone replacement.⁴² They also have an increased sense of well-being when replacement is continued. Data cited by these investigators even suggest these growth hormone deficient

adults have a much higher mortality rate. This mortality seems to be from an increased incidence of coronary artery disease, which may be mediated by increased cholesterol and triglyceride levels in growth hormone deficient patients.

Besides the beneficial effects of growth hormone on body composition and lipids, there may be effects on the immune system. Growth hormone stimulates regeneration of thymic tissue in aged subjects. It primes macrophage production of oxygen free radicals and augments granulopoiesis.⁴³ Another study showed two weeks of growth hormone treatment increased natural killer cell activity by 20%.⁴⁴

Progeria is a genetic disease with striking features that resemble accelerated aging. Very low levels of IGF-1 and high basal metabolic rates have been observed in patients with this rare disorder. A trial of growth hormone in two progeria patients resulted in increased linear growth and decreased basal metabolic rate. The authors of this study conclude that an abnormal growth hormone may contribute to the etiology of progeria.⁴⁵

Growth hormone therapy beneficially alters body composition in aging. Unlike testosterone, it promotes a favorable lipid profile. It has intriguing immune system effects. Much study is needed, however, before it can be considered a useful anti-aging hormone.

Conclusions

Anti-aging hormones are easily obtainable and highly publicized. Melatonin, DHEA, testosterone, and growth hormone have multiple systemic effects. Although serum levels seem to decrease with aging, it is not known whether these decreasing serum levels actually contribute to the aging process or are merely associated with it.

Melatonin's beneficial effects on immunity and as an antioxidant were shown in laboratory animals. Despite its attention in the media and the marketplace, its usefulness in humans will probably be limited to a treatment of insomnia and jet lag. Its production is currently unregulated and thus commercial products may contain harmful impurities. DHEA, like melatonin, is manufactured as an unregulated supplement. Although the sensation of well-being it produces may be from steroid neurotrophic effects, mechanisms for other reported effects are yet to be proven. DHEA can produce androgen side-effects in women and theoretically potentiate the risk of hormone-responsive tumors. Testosterone therapy is available by prescription for hypogonadal men and as part of estrogen replacement therapy in postmenopausal women. Male menopause is being recognized as a real entity. Long-term testosterone replacement in men needs further study but

may be limited by the risks of prostate cancer and coronary artery disease. Growth hormone, in doses that increase IGF-1 to youthful levels, may be the most promising anti-aging hormone. Much more investigation of growth hormone therapy is, of course, required before specific conclusions can be reached.

The National Institute of Aging (NIA) is currently funding large long-term clinical trials that are designed to determine the effects of boosting the levels of these hormones. In the future, hormone replacement therapy may include many different treatment combinations for aging men and women. Presently, however, despite widespread media attention and consumer forces, the potential benefits and risks of these therapies remain unproven.

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WHERE TO CALL

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The search for functionality: The role of the environment in the development and maintenance of psychopathological symptoms

by Patricia Brierley-Bowers, Ed.D.

Abstract

A brief history of applied behavior analysis is used to demonstrate the importance of functional analysis. Functional analysis is the empirical investigation of possible maintaining consequences for a specific target behavior. Case studies are used to illustrate how functional analysis may be used with individuals with developmental disabilities and possible mental illness.

The environment often plays a role in the development and maintenance of behaviors which appear pathological. Layng and Andronis,¹ through a series of case reports, demonstrated how symptoms of schizophrenia were functional in that they allowed people to avoid unwanted events or helped people solve problems. This is also true for people with developmental disabilities.

Since people may engage in apparently pathological behaviors due to factors outside of organicity, it becomes important to identify these other variables in order to adequately treat them. This article focuses on the identification of factors which maintain problem behaviors. A brief history of applied behavior analysis illustrates the role of the environment in the development and maintenance of behavior, including apparently psychopathological behaviors. Three illustrative cases are presented:

Case 1

Ms. D is a 40-year-old woman with mild mental retardation and schizophrenia. She has had Nueroleptic Malignant Syndrome (NMS) at least twice. Ms. D was in the process of being titrated on Moban in an effort to treat her psychosis without another incident of NMS. Prior to the initiation of the Moban, Ms. D was terrorized by snakes crawling on her and her belongings. After intense psychiatric intervention, Ms. D was stabilizing except for her reports about a baby riding on her back and frequent incidents of acting out the birth of a baby. The question for the psychiatric team was whether to increase the Moban and possibly increase of risk of NMS reoccurring. During observations and interviews, Ms. D would look intently into the eyes of the observer and tell her about her baby. Often she would smile when reporting this phenomenon. After interviewing the staff and conducting numerous observations, it was decided

This article is the first in a series that will explore various aspects of treating patients with developmental disabilities.

that these behaviors were not simply symptoms of schizophrenia. Ms. D maintained these behaviors after increased time with the doctors and verbalized concern of the staff.

Case 2

Mr. W is a 30-year-old man with severe mental retardation. He had been treated with Trilafon, Navane, Mellaril, Haldol, Ativan, and Zoloft for aggression (e.g., biting, hitting, and grabbing others), disrobing, disruption (e.g., throwing chairs, shoes, or other objects; tearing curtains or shades; and yelling), and self injury (e.g., head banging, biting himself, and picking his skin). Despite the use of medication over the years the problematic behaviors continued.

Case 3

Ms. M is a 32-year-old woman with moderate mental retardation who became blind at the age of 23. She has been treated with Thorazine for schizophrenia for over 13 years. Ms. M was treated for bizarre verbalizations (i.e., changing the tone or pitch of her voice and talking about death, blood, being raped, and throwing babies) and disruption (e.g., pulling her hair, touching her genitals, jumping, and turning over furniture).

Brief History

Early in the history of psychology, Pavlov² conducted studies investigating the physiological aspects of salivation in dogs. Secondary to his purpose, he noted an interesting behavioral phenomena. Pavlov observed that when cues in the environment which normally cause a physiological response (e.g., food causes salivation) are paired with cues which do not cause a physiological response (e.g., a white lab coat), they may then cause the latter to create the physiological response as well. That is, after the food and the lab coat are presented together several times, the lab coat alone will also cause salivation. This is called respondent (classical) conditioning.

Later, in the case of *Little Albert*,³ Watson and Rayner demonstrated that respondent conditioning works with humans as well. Jones⁴ then demonstrated how this connection may be broken through desensitization. However, Watson, Rayner, and Jones were unique at that time in that they worked with people. Most psychologists interested in conditioning were still experimenting with animals.

Another branch of early exploration was the role of consequences. Thorndike⁵ demonstrated how a cat would solve a problem more quickly if food was the reward. Later,

Skinner furthered this investigation using rats in mazes and pigeons pecking levers. Skinner developed the schedules of reinforcement (i.e., rate in which reinforcement may be delivered in order to shape and maintain target behavior). As early as 1957, Skinner⁶ spoke about the necessity of finding the function (the maintaining consequence) of target behaviors. This focus on the use of consequences to shape behavior is known as operant conditioning.

It was not until approximately 1960^{7,8} that operant conditioning was applied to people. Much of the early work was done with people who were institutionalized for developmental disabilities or schizophrenia. At this time, psychologists chose a provider reinforcer (i.e., anything that increases a behavior), and a possible punisher (i.e., anything that decreases behavior), and applied them to modify behavior.⁹ This technique was usually successful for a short duration. However, the target behavior often returned or new problem behaviors emerged.

In 1977, Carr¹⁰ discussed finding the function of a specific behavior — self injury. The central idea in his discussion may be generalized to any behavior. His theory is that people do things for a reason and often for more than one reason. In order to change a behavior, it is necessary to find out why a person does it and offer an alternative. By teaching a functional alternative (i.e., a behavior which results in receiving the same consequences), the old problem behavior is no longer necessary. In 1982, Iwata, Dorsey, Slifer, Bauman, and Richman¹¹ developed the technology needed to empirically investigate possible maintaining consequences for the target behaviors. This technology is known as a functional analysis. A functional analysis is accomplished by setting up conditions which may increase the likelihood that the target behavior will occur. Possible functions usually include escape or avoidance (e.g., of events, tasks, stimuli), positive reinforcement (e.g., getting attention, food, or objects), and self-stimulation (e.g., due to boredom, to obtain an endorphin high, etc.). Functional analysis has recently expanded into looking for possible antecedents or events in the environment which may trigger target behaviors.¹² Special training should be sought in order to correctly do a functional analysis.

In his review of functional analysis, Mace⁹ notes that through its utilization methodology, treatments are more likely to be effective and less restrictive. This may be true for a number of reasons. As discussed above, through a functional analysis, an alternative behavior may be chosen and then taught, which makes the target behavior unnecessary to obtain the same end result. In addition, by identifying a function for the behavior, it becomes easy to predict when a problem may occur. This predictability allows

preventive interventions to succeed. As demonstrated in the case discussions, the use of functional analysis also enables the treating professional to choose the appropriate intervention such as medication versus environmental change or skills training.

As Carr⁸ discussed in his article on self injury, behaviors may originally occur as part of psychopathology. But, the behaviors may become associated with specific consequences. Hence, the behaviors whose original function was physiological are maintained by their consequences in the environment. As Layng and Andronis¹ discuss, the function is not always apparent by the topography of the behavior. Simply saying that it looks crazy does not always make it so. It is then always necessary to question the presence of any possible maintaining consequence as well as the presence of psychopathology.

Case discussion

In two of the cases presented, psychopathology is likely present. In the other, the symptoms appear as pathological but were maintained solely by the environment.

Case 1. The function of Ms. D reporting a baby riding on her back and acting out the birth process was attention seeking (positive reinforcement). Treatment involved noncontingent attention by favored staff and ignoring of the problematic behaviors. Within months, Ms. D's rate of reporting was reduced to near zero. In this case, apparent hallucinations were both psychopathological and behavioral. The treatment of choice was a combination of medication and behavioral treatment.

Case 2. Through interviews with the staff, several hypotheses were developed regarding Mr. W and the possible function, including escape from noise and time of day (only in the afternoon). A functional analysis was designed specifically to address these hypotheses. Neither were found to be significant. The analysis suggested that Mr. W engaged in target behavior only when someone spoke to him. The intervention package included desensitization to being addressed. Prior to beginning the treatment package, he was taken off all medications. Since the intervention there have been no instances of target behaviors and no injuries for over six months. In this case, medication did not sufficiently treat the problem, but through careful analysis, a behavioral treatment plan was successful.

Case 3. A functional analysis was conducted while Ms. M was on the Thorazine. No hypothesis could be formulated because Ms. M engaged in the target behaviors in all conditions (at least 50% of the time) and was more likely to do so in two conditions (100% of the time). It was decided that the medication was not working for Ms. M. The Thorazine was discontinued and a new analysis con-

ducted. In this analysis, the target behaviors occurred in all conditions nearly 100% of the time. Risperidone was then initiated. The target behaviors then decreased over time to near zero. In this case, the environment played no significant role, but the analysis helped demonstrate the effectiveness of the appropriate medication.

The cases presented illustrate how apparent symptoms of psychopathology may be maintained by consequences. Each of the cases demonstrates the impact of the environment on behaviors which appeared to be psychopathological. In the first case, the analysis suggested a dual function. The reports of the baby and pregnancy were due to organic sources (schizophrenia) and environmental factors (positive reinforcement). In the second case, the violent behaviors were clearly maintained by environmental influences (escape, avoidance, and/or respondent conditioning). And in the last case, the bizarre verbalizations were maintained by physiological influences. However, in each case, the analysis or assessment of possible maintaining consequences led to the most effective, least restrictive intervention.

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Dealing with uncompensated care: A hospital board perspective

Providing care to all regardless of ability to pay is key to Liberty Medical Center's mission; it is also key to Liberty's financial challenge. At Liberty Medical Center, over 14% of the care rendered is uncompensated. The cost of uncompensated care is Liberty Medical Center's second highest expense, behind only salary and wages. As a percentage of its revenues, Liberty's uncompensated care burden is the highest in Maryland. While for Liberty \$1 out of \$7 of care is uncompensated, on a state-wide basis, the average is approximately \$1 out of \$11, and there are providers in the Baltimore-metropolitan area with rates of uncompensated care below \$1 out of \$50.

In many states, Liberty's uncompensated care burden would long ago have resulted in its demise. Fortunately, the Maryland rate system affords Liberty the opportunity to transfer the cost of uncompensated care to its paying patients, but the result is that their bills are more than 14% higher than the cost of services actually rendered to them. Unfortunately, this cost transfer causes a severe competitive disadvantage because Liberty's rates to paying patients are substantially higher than they ought to be.

Regulators, insurance companies, managed care organizations (MCOs), and the general public have all become extremely conscious of health care costs. Insurers and MCOs force health care providers to lower charges by directing plan members to the lowest cost providers and prohibiting members from using a higher cost hospital even if a member lives in the community where that hospital is located. For patients, this can be disadvantageous because they and their families may be forced to travel farther for care and to receive care from physicians and facilities unfamiliar to them. For hospitals with high uncompensated care such as Liberty, this redirection of patients reduces census; it also reduces the proportion of paying patients to non-paying patients, thereby further raising costs for the remaining paying patients.

There is only one solution — reduce costs. Liberty's board and administration have been pursuing this

often painful task for several years, with the attendant lay-offs, reorganization, and restructuring. Staffing, length of stay, use of ancillary services, and other costs at Liberty have been brought to, or very close to, industry norms.

For urban hospitals, however, the possibility of reducing costs ultimately is limited by the extent to which they cannot reduce the amount and cost of uncompensated care.

Presently, the biggest single factor in the amount of uncompensated care that a hospital faces is location. Inner-city hospitals have more uncompensated care because they are located in the midst of a population that has less than average health care coverage from private insurance, employees, or government programs. Liberty Medical Center's primary service area is made up of diverse neighborhoods located within the five zip codes surrounding the hospital. In one of its primary zip codes, almost 40% of the population has a household income below poverty level. This population has dramatic health care needs, almost no private insurance, and a high unemployment rate. In addition, a large segment of the population is not eligible for Medicare or Medicaid. Because of an inability to pay, the population has not attracted adequate physician care. There are few physician offices and other health care outlets for this population. Consequently, when these individuals become ill, their illness often remains undiagnosed or untreated until it reaches a high level of acuity.

Liberty's board of directors established a two-pronged approach to dealing with the problem. First, the board is committed to increasing primary and specialty care available in Liberty's service area in order to reduce acute hospital care needed. Second, the board is committed to working aggressively to redistribute, through various payment system devices, the burden of financing uncompensated care.

Liberty's strategy of increasing available primary care has been executed by placement of primary care facilities in the community. Liberty created Park Circle Medical Associates, a multi-physician practice with

offices at the hospital and at several satellite locations, to make primary care, as well as specialty care, available in locations proximate to the patient population. Liberty's board also created Liberty Medical Care — which operates several physician offices in the community, assuring the continued availability of primary care in these locations. Liberty also entered into a partnership with a number of physicians known as Seton Medical Associates to establish one of the largest physician practices in Liberty's market area. Through each of these operations, Liberty has been expanding and insuring the availability of non-hospital care in an effort to reduce uncompensated care by providing interventions at earlier stages of illness and by increasing the preventive health care individuals receive.

A major new initiative also intended to upgrade community health status is the Urban Medical Institute. This innovative program is located in a new facility adjacent to Liberty Medical Center. The program includes wellness, health prevention, and research components. It will focus efforts on the diseases which are most endemic to the community such as diabetes, hypertension, and pulmonary diseases. The program is built on the premise that early detection and early intervention will create a healthier population, requiring less hospitalization and less intensive health care services. For example, the diabetes care management program will manage the patient affected with diabetes and, through education and nutritional assistance, keep that patient from developing the more acute symptoms associated with the disease. The Urban Medical Institute also will include significant efforts at early detection through screening and prevention programs for breast cancer, prostate cancer, and hypertension. It is the board's philosophy that these programs are good both for the health and welfare of the community and also, through the reduction of the utilization of its more costly inpatient programs, for the financial health and welfare of Liberty.

In addition to reducing the levels of acute care required, and thereby reducing the most expensive uncompensated care, Liberty's board of directors also

focused effort on effecting a redistribution of the financial burden of uncompensated care. Working through the Maryland Hospital Association, the various regulatory agencies, and the state legislature, Liberty has been very active in seeking modifications of the current rate-setting methodology to help equalize the rate burden of uncompensated care. Liberty has been an advocate of having hospitals with lower uncompensated care burdens include in their rates more of the uncompensated care burden, with a transfer payment to hospitals having higher uncompensated care burdens, thereby enabling the latter hospitals to reduce rates to more competitive levels. The Maryland Health Services Cost Review Commission recently adopted regulations to implement this concept.

The board of directors of Liberty has consistently viewed uncompensated care as a critical aspect of its mission. Liberty Medical Center was created to serve the underserved and to ensure that all members of its community receive quality health care regardless of their ability to pay. Liberty has consistently pursued this mission and will continue to do so. At the same time, the board has recognized that the hospital cannot thrive—or even survive—solely by treating a non-paying population. In order to attract and treat the population whose medical care is paid for by cost-conscious insurers and MCOs, the board realizes that Liberty's rates must be competitive, and to that end, the board has vigorously pursued its two-pronged approach. Although the path has been difficult and the financial burden heavy, the prospects appear to be improving. As initiatives such as the Urban Medical Institute are fully implemented, and as the regulations for rate relief are put into place, Liberty anticipates the day when it will successfully be reaching into its community to improve health status, reduce the need for acute hospitalization, and have rates which allow it to be competitive with other urban health care providers for the patients of cost-conscious third-party payors.

DAVID C. DANEKER AND LAWRENCE RYCHLAK

Mr. Daneker is past chairman of the board of Liberty Medical Center and Mr. Rychlak is the chief financial officer of Liberty Medical Center. ■

MEDICAL HISTORY

The growth and metamorphosis of Provident Hospital and Free Dispensary into the Liberty Health System, Inc.

Provident Hospital on Liberty Heights Avenue is a remarkable institution. Like the mythical Phoenix that recreated itself from its own funeral pyre, the hospital has undergone two resurrections. In 1973, after moving to Liberty Heights Avenue, it was placed in receivership. After a subsequent closure in 1985, a second resurrection occurred when it joined the Lutheran Hospital and became the Liberty Medical Center.

The area in which these two institutions arose was in the west central part of Baltimore (Figure 1). This was the "Sugar Hill" area, containing the blocks along Pennsylvania Avenue. The "Bottom" was situated below Dolphin Street where Orchard and Biddle Streets are located.¹

Residential, two-story brick homes lined the streets and alleys below Dolphin Street. At a later time, three-story row houses with marble steps were built up to North Avenue. Houses along Biddle Street were narrow and crowded, so disease spread easily. This was the area of the notorious "Lung Block," so called because of its high incidence of tuberculosis. A satisfactory sewage system was lacking. Overcrowding, coupled with despicable environmental conditions, led to elevated rates of morbidity and mortality in this area.

A mixed population lived in this crowded region in the 1890s. First generation African-Americans living in the area were descendants of a community of free men and women who had lived there before the Civil War and the families of slaves coming from rural Maryland and the upper South. An enormous contingent of white European immigrants came through the port of Baltimore and also settled here. These individuals were in fierce competition for jobs in household labor – servants, butlers, carriage drivers, yard workers, nurses, cooks, and chambermaids.

Baltimore was a growing city with about 455 000 people.² The majority were white (84%) as compared to black (16%). A major indication of the poor status of blacks was the death rate: 30.15 per 1 000 compared to the white death rate of 20.98 per 1 000. The eleventh ward, just west of

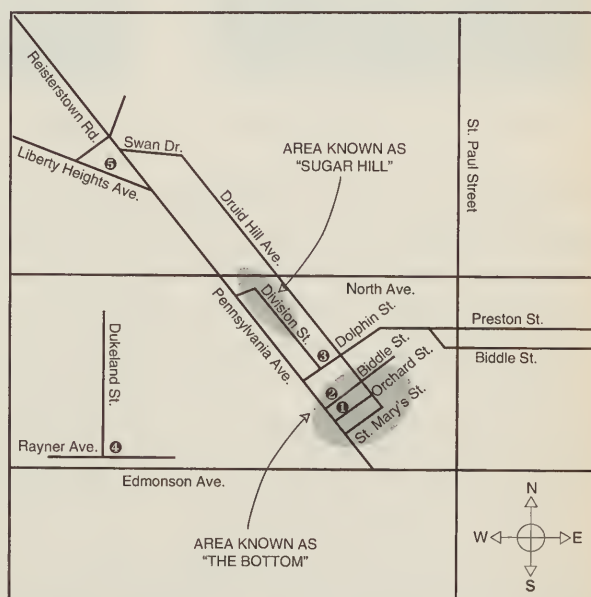


Figure 1. Hospital locations. 1 - Orchard Street, 2 - Biddle Street, 3 - Division Street, 4 - Rayner Avenue and Dukeland Street, 5 - Liberty Heights Avenue.



Figure 2. William T. Carr, Sr., M.D.



Figure 3. Robert L. Jackson, M.D.

Dolphin Street, had a population of about 21 000, with blacks slightly outnumbering whites (52% to 48%). The rates of incidence of cholera infantum, diphtheria, typhoid fever, and tuberculosis were greater than at any other city ward.

The Provident Hospital and Free Dispensary originated in 1894 in a house at 419 Orchard Street, in a neighborhood where a small number of hospitals serving the white population were already located. At that time, the admission of black patients to white hospitals was either restricted or prohibited. Recognizing the need for black people to have a place for care and treatment, a small group of doctors started the hospital. The dream of the founders was summarized in the first annual report of the hospital: "To provide an institution where people of color may be attended by physicians of their own race; secondly, that colored physicians may have an opportunity to develop themselves along the line of their specialties, and thereby become proficient in them; and thirdly, that there may be established a well organized training school for nurses, where young ladies may obtain instruction pertaining to their calling." This group of founders included Drs. William T. Carr, Sr. (Figure 2) and J. Marcus Cargill.

The early doctors served both as members of the board of trustees and as the hospital staff. Among the group were three black physicians who were admitted as members of the Medical and Chirurgical Faculty of Maryland in the

1880s.³ (Further membership by African-American physicians was denied until 1950). The three Med Chi members were Whitfield Winsey, M.D., who was born in Baltimore in 1842, became a pupil of Dr. John R. W. Dunbar (a professor of surgery at Washington University), and received a medical degree from Harvard in 1877; Reverdy M. Hall, M.D., also a Baltimorean, who graduated from Howard as a medical doctor in 1872 and was dean, and later director, of the hospital; and William H. Thompson, M.D., who was born in Yonkers, New York in 1849 and received his doctorate degree at Howard University in 1872.

The hospital's first home on Orchard Street soon proved to be too small. In 1896, the hospital was moved to 413 West Biddle Street where it could house 30 beds. An adjoining building was added for a nurses' training school.⁴⁻⁸

The institution was the center of care for a number of black physicians who were intensely interested in its welfare. Early practitioners also included Drs. R.D. Grant Scott, Harry F. Brown, and Charles Fowler. At a later period, Dr. Robert L. Jackson (Figure 3) was the first resident surgeon and in 1928, became the medical superintendent. He rose to be the chief of surgical services when Dr. George G. Finney went into the Army in 1942.

Although Provident Hospital was mainly created by black doctors and the black Baltimore community, many white

physicians made contributions to the care of its patients. After Union Memorial Hospital moved from Division Street to new quarters, the suggestion was made that the black population of Baltimore raise \$75,000 to buy the old building. When the purchase of the hospital was previously contemplated, Dr. John M.T. Finney, Sr., served as the committee chair and obtained foundation support, state funds, and generous gifts from the white community. Within a month, this money, added to the \$165,000 produced by the black population, led to the dedication of the hospital on Division Street (Figure 4). At that time, Dr. Finney, Sr., also pledged that he and other white



Figure 4. Provident Hospital on Division Street

doctors would be willing to be part of the staff.⁹ After Dr. Finney, his two sons, John, Jr., and George, Sr., (**Figure 5**), and a grandson, George, Jr., continued to offer medical help on Division Street.

The institution opened on October 15, 1928, with Dr. William T. Carr as superintendent. Unfortunately, it was at the start of the great economic depression, and at the end of 15 months of operation, the hospital had a deficit of \$30,000. With the cooperation of all concerned, a sum of \$300,000 was raised and the debt retired.

At about this time, 16 African-American physicians were appointed to the visiting staff with privileges for the treatment of private patients. The American Medical Association approved a plan for training six interns in a general rotating program. In addition, approval was gained for residencies in pediatrics (two years), general surgery (three years), and anatomic pathology (one year). Provident was one of only five black hospitals in the United States that gave specialty training. Over the years, service and care was provided for about 250 000 people.

The School of Nursing

Started on Biddle Street in 1895, the Provident Training School for Nurses was the first and only institution in this area to offer African-American women the opportunity to train as nurses. Dr. Carr founded the school with the help of Lena V. Ashyon, a graduate of Freedman's Hospital in Washington. The school was given official recognition in 1926. The name was first changed to the Provident Hospital School of Nursing in 1950. After Dr. Leonard F. Fuld and his sister honored their mother by establishing the Helene Fuld Health Foundation, which offered full scholarships for student nurses, the name of the school was changed in 1963 to the Helen Fuld School of Nursing of Provident Hospital. In 1974, charge of the school was transferred to Coppin State College so



Figure 5. George G. Finney, Sr., M.D.

that a bachelor's program in nursing could be provided.

An interesting sidelight of the School of Nursing was the establishment of the Gamma Chapter of the Chi Eta Phi Sorority of black nurses in 1946, a time when black nurses encountered difficulty in obtaining employment. Created by 14 graduates of Provident Hospital and 2 from Freedman's Hospital in Washington, the sorority recently celebrated its 50th anniversary.

Further Growth of the Hospital

A modest expansion with the acquisition of some new equipment was undertaken by the hospital in 1951. Community efforts raised \$300,000, enabling the addition of 12 beds. Drs. Bernard Harris (**Figure 6**) and I. Bradshaw Higgins (**Figure 7**) were of immeasurable help during this period.

The later 1960s brought new hope for better hospital quarters and the opportunity to replace the outmoded equipment in the Division Street structure. Thus, a modern building on Liberty Heights Avenue was occupied in 1970.

Unfortunately, financial difficulties were present from the outset. Problems included a low rate of occupancy and a lack of expected community support. A receivership under the guidance of George L. Russell, Sr., was established in 1973, and he was able to restore financial stability after a short period of reconstruction.



Figure 6. Bernard Harris, Sr., M.D.



Figure 7. I. Bradshaw Higgins, M.D.



Figure 8. Lutheran Hospital

By 1976, Provident Hospital was well on the road to recovery although some of its internal problems were still present. Perhaps the greatest concern was the need for more admissions. The integration of African-American doctors and patients into previously Caucasian-only hospitals siphoned many potential patients from Provident Hospital. An ever-present obstacle in reaching financial stability was the surrounding low-income community.

The next few years saw many positive clinical events at the hospital. Surgical and obstetrical residents and summer students were rotated from Meharry Medical College. The operation of a hypertension program with numerous outreach facilities was facilitated by a grant-in-aid. A cancer-screening clinic, fundamentally supported by the Morris Goldseker Foundation of Maryland, Inc., was an entirely free program for individuals in the hospital area.¹⁰ The clinic educated the community about the benefits of receiving an annual physical examination. In the clinic's first year, 5 carcinomas were found in 1 050 individuals screened, providing another indication of the high morbidity rate for cancer among blacks.

A Sickle Cell Disease Program was similarly financed.¹¹ Patients were offered treatment, counseling, and hospitalization. Screening was done in a target area of about 200 000 blacks. Almost 97% of the individuals screened were black, and the total who had electrophoretic studies of the blood was 39 510. In those studied, some type of hemoglobinopa-

thy was demonstrated in almost 11%. HbSS (Sickle Cell Disease) was found in 0.09% and HbAS (Sickle Cell Trait) in 7.85%. Other types of hemoglobin, including HbAC, HbAF, HbSC, accounted for the other 3.03%.

An offshoot of this study was the first demonstration of a 6th isoenzyme of lactate dehydrogenase occurring regularly in HbSS.¹² A later study of the same system in amniotic fluid of healthy African-American women showed only the five fractions occurring regularly in blood.¹³ An "extra" isoenzyme, such as that encountered in placental samples, was not found.

Another unique piece of investigation involved the testing of 722 African-American hospital patients for the Duffy antigens.¹⁴ It was already known that a racial variant existed; Caucasians always showed a positive reaction to antigens, while the value of antigens was lower in those of African descent. In the largest population ever examined in the United States, this landmark investigation revealed that 36.28% of the group tested positive for Duffy antigens, indicating a strong admixture of the races.

In 1985, financial difficulties led to a fatal hospital crisis. A low occupancy rate, with its consequent problems of poor cash flow, led to the closing of the hospital once more. In 1986, the hospital reopened, having merged with Lutheran Hospital to form the Liberty Medical Center.



Figure 9. Liberty Heights Medical Center

Lutheran Hospital (**Figure 8**) also had an interesting beginning.^{15,16} In 1873, William Rayner, a prominent Baltimorean, donated land at Rayner Avenue and Dukeland Street upon which to build the Baltimore Hebrew Orphan Asylum. The institution, chartered in the previous year, had 125 students. When the asylum moved to Mount Washington in 1923, West Baltimore General Hospital, underwritten by area doctors and businessmen, moved into the structure. At this time, a nurses' quarters, maternity wing, and power plant were added. By 1939, the hospital contained 100 beds and 20 bassinets. After World War II, the Lutheran Home and Hospital was organized upon the existing structure.

Recent Growth of Liberty Medical Center

Again, financial stability was achieved, although Liberty Medical Center still cares for the indigent, underinsured, and underserved people of northwest Baltimore. The creation of the Urban Medical Institute by the Liberty Health Care System seeks to reduce hospital admissions by a judiciously managed health care program. Focusing on wellness, education, research, and disease prevention, a reduction in morbidity and mortality is being sought for this population, which traditionally has a high percentage rate of Medicare and Medicaid assistance.

The effort to provide multiple modalities of managed care has resulted in further expansion of Liberty Health System's capabilities. Formation of the Community HealthCare Network of Baltimore resulted in a partnership with Bon Secours Health Corporation. A secondary alliance, the Liberty membership in the Metropolitan Baltimore Community Health Alliance, includes a partnership between community-based health centers and hospitals throughout Baltimore. The accent of these organizations will be the provision of primary and preventive care. Acute and specialty services will be obtained by referral.

The tiny original hospitals have grown to an institution of 282 licensed beds (**Figure 9**). The acceptance and proper utilization of managed health care, with particular emphasis on the early treatment of chronic disease and the adoption of preventive measures, is designed to carry the hospital into the next millennium.

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Early Maryland physicians and the first presidential election

	FIRST SESSION										
	George Washington	John Adams	Samuel Huntington	John Jay	John Hancock	Robert H. Harrison	George Clinton	John Rutledge	John Milton	James Armstrong	Edward Telfair
New-Hampshire	5	5									
Massachusetts	10	10									
Connecticut	7	5	2								
New-Jersey	6	1									
Pennsylvania	10	8			2						
Delaware	3		3								
Maryland	6				6						
Virginia	10	5		1	1		3				
South-Carolina	7				1		6				
Georgia	5								2	1	1
	69	34	2	9	4	6	3	6	2	1	1

RECAPITULATION of the VOTES of the ELECTORS		
His Excellency George Washington	-	69 Votes
The Honorable John Adams	-	34
The Honorable John Jay	-	9
Robert H. Harrison, Esquire	-	6
John Rutledge, Esquire	-	6
John Hancock, Esquire	-	4
George Clinton, Esquire	-	3
Samuel Huntington, Esquire	-	2
John Milton, Esquire	-	2
James Armstrong, Esquire	-	1
Edward Telfair, Esquire	-	1
Benjamin Lincoln, Esquire	-	1

On motion,
ORDERED, That a message be sent to the Senate, to inform them that it is the desire of this House that the notifications of the election of the President and Vice-President of the United States, should be made by such persons and

⁴The votes with related documents are in Election Records: Electoral votes, SR, DNA.

Figure 1. List of the votes of the electors of the several states in the choice of a president and vice-president of the United States in the 1789 election.

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When President William Jefferson Clinton was formally re-elected by the Electoral College during a ceremony at a joint meeting of Congress on January 9, 1997, a tradition of over 200 years was again carried out as required by our Constitution. Completed and signed on September 17, 1787, Article II, Section I of the Constitution created the college as an indirect system to elect our president and vice-president.

The Electoral College has existed since the beginning of our political system. On March 4, 1789, the date set aside by the first federal Congress to certify and count the electoral ballots, a quorum could not be formed in the House. A total of 13 members were seated; none were from Maryland. By April 1, 17 additional representatives had arrived in New York, and a quorum of the House was finally achieved. Two members from Maryland were present. A third, Daniel Carroll of Montgomery County, was seated on that day.¹

Senator Richard Henry Lee of Virginia took his seat in the Senate Chamber on April 6, and his presence constituted a

quorum for a joint meeting of the Congress.² A message was sent to the House informing the Speaker that a quorum of the Senate was present. John Langdon of New Hampshire was elected president pro tempore of the Senate for the sole purpose of opening the certificates and counting the votes for the electors of president. In the presence of the House and Senate, the ballots were counted and recorded in the Senate Journal (Figure 1).

In the first presidential election in 1789, two of the eight Maryland electors were physicians (Figure 2). They were Dr. Philip Thomas of Frederick County, a founding father and the second president of the Medical and Chirurgical Faculty of Maryland, and Dr. William Matthews of Cecil County. Both had been selected as candidates by the Federalist party and were later elected by the state voters on a general ticket.

The entire federalist slate of eight electors, two senators and six representatives, was easily elected because it ran in a statewide general election, rather than by districts. At that time, the Federalist party was the only organized political

group in the state. Electors were prominent members of the party who did not hold any political office. Various factions that made up the opposition were known as "Antifederalists."

In the general state election, Dr. Thomas finished seventh with 5 456 votes, and Dr. Matthews was eighth with 5 291 (**Figure 3**). The Federalists were especially strong in the western counties. Dr. Thomas, representing Frederick, Montgomery, and Washington counties, met little opposition. Washington County voted unanimously for both the electors and members of Congress, and no "anti" votes were recorded. Dr. Matthews represented the counties of Cecil, Kent, Queen Anne's, and Talbot. His native county, Cecil, also voted unanimously for all Federalist candidates.

George Washington was the only person considered for the presidency of the new nation, and each elector had two votes for president as prescribed by the Constitution. The candidate who received the largest number of votes by the college became the president, and the runner-up became the vice-president.³ This system, eventually found to be unworkable, was changed in 1804 by the 12th Amendment. The electors thereafter cast one vote for president and one for vice-president.

Washington should have received a total of 91 electoral votes from the 13 original states. Actually, he received 69 votes. John Adams received 34 votes and became vice-president. Rhode Island, with three votes, and North Carolina, with seven, had not yet ratified the Constitution. New York's eight votes were lost because they were not cast on time. Two electors from Virginia and two from Maryland failed to vote. One elector from Maryland, George Plater of St. Mary's County, remained at home with an acute attack of gout, and the second Maryland elector, William Richardson of Caroline County, was unable to reach Annapolis because of ice on the rivers and Chesapeake Bay.^{4,5}

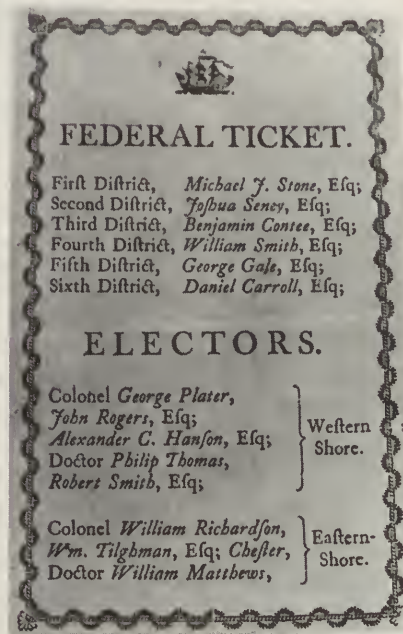


Figure 2. Broadside of Maryland federal electoral ticket, 1789.
 Reprinted with permission, Maryland Historical Society, Baltimore.

When the ballots were finally counted in New York on April 6, 1789, the six electors from Maryland gave each one of their votes to Washington, and their other six presidential votes went to Robert Hanson Harrison, a "favorite son." The electors from other states scattered their second vote for the various other 11 presidential candidates. Harrison would have made an excellent vice-president; an experienced lawyer, he had been a friend and associate of General Washington during the Revolutionary War. He was one of the general's most trusted aides-de camp and personal confidential secretaries. After the war, Harrison was appointed chief judge of the General Court of Maryland after turning down an appointment to the Supreme Court because of poor health. He died at Port Tobacco on April 2, 1790.⁶

Dr. Philip Thomas' connection with George Washington did not stop with the presidential election. He later served the federal government as one of its officials in Western Maryland and was associated with Washington on several other occasions.

During his first term, President Washington was chiefly occupied with the organization of the new government. The formation of a revenue system was an early administration duty, and in a letter to the secretary of the treasury dated March 15, 1791, he outlined how revenue was to be col-

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ELECTORS FOR PRESIDENT.

	St. Mary's.	Calvert.	Queen Anne's.	Talbot.	Kent.	Cecil.	Annapolis.	Anne Arundel.	Baltimore Town.	Baltimore.	Harford.	Dorchester.	Somerset.	Frederick.	Charles.	Prince Georges.	Washington.	Montgomery.	TOTAL.
Chancellor John Rogers	110	100	804	80	884	178	519	314	464	510	843	745	444	111	312	163	199	780	7885
Col. George Plater.	114	128	258	48	858	100	512	201	453	404	898	728	441	112	312	158	199	787	7878
Col. William Tilghman	84	114	106	51	808	300	519	100	117	209	470	142	304	118	308	98	110	76	5740
William Richardson.	90	110	106	48	854	100	519	100	117	209	470	142	304	118	308	98	110	76	5740
Alexander C. Hanson.	90	110	106	48	854	100	519	100	117	209	470	142	304	118	308	98	110	76	5740
Robert Smith, Atty.	90	110	106	48	854	100	519	100	117	209	470	142	304	118	308	98	110	76	5740
Dr. Philip Thomas	92	181	100	89	204	178	519	88	118	370	470	142	308	109	311	69	128	790	5881
Dr. William Matthews	100	191	108	27	847	174	519	94	110	307	474	142	308	89	311	1	137	787	5881
Jeremiah T. Chase.	12	8	90	20	41	170	872	398	879	113	230	60	80	80	80	80	80	80	2878
Charles Ridgely, of Wm	11	8	84	18	37	85	845	281	877	683	289	78	1	2	1	1	1	1	2190
John Sency	18	18	88	40	88	108	846	281	877	683	289	78	1	2	1	1	1	1	2190
James Shaw	18	4	80	7	80	92	846	219	858	617	240	68	9	9	9	9	9	9	1660
Henry Waggoner	4	4	4	4	4	60	889	218	850	604	4	30	9	74	1	1	1	1	1241
Lawrence O'Neale	6	9	10	10	80	19	10	10	821	590	204	1	1	1	1	1	1	1	718
Thomas Johnson	101	896	845	29	1	101	896	845	29	1	1	1	1	1	1	1	1	1	125
William Thomas, Jr.	80	7	80	8	1	80	8	1	80	8	1	80	8	1	80	8	1	80	83
William Pace.	83	83	83	83	83	83	83	83	83	83	83	83	83	83	83	83	83	83	70
Nathaniel Hamoy	105	105	105	105	105	105	105	105	105	105	105	105	105	105	105	105	105	105	105
John Dene.	187	187	187	187	187	187	187	187	187	187	187	187	187	187	187	187	187	187	187
Miss Rawlings.	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
John A. Thomas.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
George Deni.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

Female Excess.

Figure 3. List of the votes for electors from Maryland, by county, in the first presidential election of 1789.

Source: Scharf. *History of Maryland*. Vol. II. 1879:549.

lected on distilled spirits, both imported and domestic. Philip Thomas was appointed surveyor (inspector) to collect an excise tax on whisky in Western Maryland. His salary was \$450.00 plus a commission of 1%.⁷

There were many private or family stills in his area and the tax became a burden and was very unpopular. Commercial distillers were also hurt by the levy and began a vigorous protest. The so-called "Whisky Insurrection of 1794," which occurred in western Pennsylvania and adjacent Maryland, brought about a change in the law exempting the small stills.

On October 20, 1790, at the request of his friend, General Otho Holland Williams, Dr. Thomas, together with Colonel Elie Williams, was the host for President Washington's inspection of Williamsport. The banks of the Potomac, at the mouth of Conococheague Creek, were at that time under consideration as a site for the nation's capital.⁸

When President Washington passed through Fredericktown on June 30, 1791, on his way to Philadelphia from his southern trip, he was greeted by many prominent Federalists, including Dr. Thomas and his son, John Hanson Thomas.⁷

Dr. Matthews also continued his political career after the first presidential election. A third-generation physician, he lived and practiced in Cecil County and had represented his native county in the lower house of the State Assembly from 1786 to 1789. He was elected to the U.S. House of Representatives from 1797 to 1799. He later sold 1 116 acres, including his farm of 574 acres, and moved to Kent County. He died in 1808 at age 53 and unmarried.⁹

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Med Chi's presidents: a series of historical vignettes

An institution's continuing growth depends largely upon the caliber of people who serve as its leaders. Many of Med Chi's presidents were among the best practitioners of their times. This series of historical vignettes will provide insight into the capabilities of these leaders.

PRESIDENTIAL TRIVIA

A review of the 147 men who have held the office of Med Chi president reveals the following facts:

- ◆ The oldest man to be elected president was Samuel P. Smith at age 83, the youngest was G. Milton Linthicum at age 39. The average presidents' age was 50, but in the last two decades, this has risen to 57.
- ◆ Three families have included presidents. Peregrine Wroth was president in 1849, and a distant relation with the same name was president in 1928. A much closer relationship existed when the Baer brothers were elected. Michael Shellman Baer was president in 1852 and Joseph Shellman Baer followed in 1855. Aaron Friedenwald was elected in 1889, and his son, Harry, in 1923.
- ◆ Nineteen men have served as president of both the Baltimore City Medical Society and Med Chi. In all instances, election by the city society came first.
- ◆ Philip Thomas, a founder, was in office longest, from 1801-1815. His duration in office was closely followed by that of George C. M. Roberts (1859-1870).
- ◆ Some presidents were elected again after an interval. Joel Hopkins was in office in 1841 and again in 1858; Robert S. Steuart in 1848 and 1850; and Samuel Chew in 1879 and 1898.
- ◆ Unfortunately, four physicians died in office. George C.M. Roberts did not finish his term in 1870; Jacob E. Michael in 1895; Herbert Halan in 1923; and Thomas B. Johnson in 1926.
- ◆ In 1983, Roland T. Smoot, M.D., was the first African-American president elected.

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"Surfing the net" leads to board inquiry

The division of drug control received an anonymous phone call claiming that a Maryland physician was prescribing medications for a person residing in another state. The caller stated that the physician had "no relationship with the patient other than through the Internet." They apparently met in a chat room encounter. Medication obtained in Maryland by utilizing a prescription made out in a false name had been mailed to the Internet contact. The Board of Physician Quality Assurance (BPQA) was asked to investigate the allegations of inappropriate prescribing and dispensing of medication and "uttering a false prescription."

A BPQA investigator contacted the physician, a third year resident, and inquired about the circumstances surrounding the allegations. The resident admitted to extensive telephone contact with a person encountered on the Internet. The resident felt the comments made by the Internet contact appeared to indicate a profound hopelessness and depression. Interpreting the Internet postings as a cry for help, the resident reached out to this person. Subsequently, the resident provided the Internet contact with a work telephone number, and a series of long distance conversations ensued. The Internet contact confided life circumstances, including an unhappy marriage, anticipation of an unwanted child, and inability to sleep or concentrate. Suicide was being contemplated.

The resident encouraged the person to seek medical treatment, but the suggestions were rejected. Monetary concerns and a desire to keep the spouse from finding out about the depression were the excuses given. After many conversations, the resident consulted colleagues about the situation. They allegedly recommended the resident offer medication for depression. Subsequently, the resident filled a prescription using a false name and mailed the medication to the Internet contact. Because insurance was not involved, the resident did not feel filling the prescription in a false name was a problem. In subsequent weeks, the resident prescribed further medications and dispensed them through the mail. Almost daily telephone calls continued until finally, despite pleas that the resident remain the doctor, the resident was able to convince the Internet contact that the services of a psychiatrist were needed. The resident did not bill for services. In fact, since the Internet contact called collect, the resident had huge phone bills.

The board requested any medical records that recorded the events that had occurred or documented the medications dispensed. The resident said the scraps of paper documenting the encounters were misplaced. In a written response to the board, the resident stated that the words of a psychiatry preceptor were the reason for trying to help this

person: "For every patient who committed suicide there was someone to whom they cried out to for help. The majority of the time, they just weren't heard.' I felt that I was this 'someone' and I couldn't be 'deaf'."

■ **This case was referred to the full board with the recommendation that the physician be charged with inappropriate prescribing, failure to maintain a medical record, and uttering false prescriptions. If you were a member of the BPQA, what would you have done with this case?**

After discussion, the BPQA asked the resident to attend a case resolution conference. If this offer were declined, the board would proceed with charges. Subsequently, the resident met with members of the board who tried to understand the resident's appreciation for the provisions of the Medical Practice Act that had been breached. The resident was very proud of the influence that being a physician generates among family members and social contacts. There was no appreciation of the issues of prescribing for a person the physician had not personally examined and for whom no medical record was maintained. The resident viewed prescribing using a false name as being expedient.

■ **Members of the case resolution conference concluded that the resident was "clueless" but had acted out of compassion. There was great concern that the resident, if not apprised of the requirements applicable to medical prescribing, would experience more difficulties in the future. Now, what would you do with the resident?**

The resident was advised to voluntarily enroll in a medical ethics tutorial and to return in three months. The tutor

graded the resident's performance in grasping the issues surrounding appropriate boundaries between physician and patient as adequate. The reasons for maintaining a medical record were reviewed. Concerns about prescribing for friends and family members were explored. Subsequently, the resident returned to the case resolution conference. The resident was asked to comment about the tutorial experience: "I'm grateful that you made me do this. I learned a lot. I will be a better physician as a result of this." Members of the BPQA agreed to close this case without further action.

Comment by Cheryl Winchell, M.D., Secretary/Treasurer, BPQA:

It is unusual to have a case where the physician's naiveté is so clearly the reason for being before the board. This resident had only been licensed a few weeks. The BPQA was anxious to find a way to get this physician on track without putting a permanent blot on his or her record. Essentially, by entering into an agreement for a corrective action, the resident was spared a public charge, and the board was able to carry out its mandate of protecting the public.

The tutorial the resident was asked to take involved significant time and expense. The BPQA has found that this one-on-one approach is likely to succeed where a didactic general course designed for continuing medical education does not. When we specify an "ethics course" as part of a corrective action or a public disciplinary order, the physician is expected to explore his or her

individual issue in depth and essentially work with the tutor until the tutor is satisfied that "he or she gets it."

This physician fell into the "only I can help this patient" trap. This same mind set has led many physicians to the BPQA. In a previously published case dealing with "Dr. A" prescribing narcotics to an addict (*Md Med J* 46;1:31), there was a similar dynamic. When a patient refuses to do what is appropriate, the physician needs to recognize his or her limitations in the ability to help. Often, excessive personal involvement leads to rule breaking and professional judgment lapses.

So, where is a young physician supposed to learn "the rules" of professional behavior? Most of us assume our teachers know them. Sadly, this may not be the case. The resident claimed that when colleagues were consulted they advised the patient be treated for depression. Did they know the patient was a phone contact? Would they have agreed the resident should mail medications to the patient, absorb the expense, and falsify a prescription? This is not to say physicians should not be compassionate or they should not offer free care. But when a physician's ego outruns common sense, bad consequences may ensue.

BPQA intends to require new licensees to undergo orientation to Maryland laws that apply to medical practice. We hope to give new licensees a better chance of avoiding disciplinary action by explaining how the licensing body has interpreted and applied the laws to their predecessors. When acting proactively, the board has its best chance to fulfill its charge of protecting the public. ■

The opinions expressed in this column are those of Dr. Winchell and are not endorsed by the BPQA.

What Your Patients

MAY BE READING

- **No Excuse For Pain**
Doctors have the means at hand to relieve the suffering of millions of Americans. Why aren't they doing it?
U.S. News & World Report, March 17, 1997
- **Is Mental Illness Catching?**
It may sound incredible, but there's evidence that psychological conditions such as depression, obsessive-compulsive disorder and schizophrenia can be caused by strep throat, the flu and other illnesses.
American Health for Women, March 1997
- **The Best News Yet About The Pill**
The latest studies and the best experts conclude it's safer than ever.
Glamour, March 1997
- **Iron – Why Growing Kids Need it Most**
This mineral is essential to your child's development, yet nearly half of today's kids don't get enough. Here's how to ensure your child's diet delivers.
Child, March 1997
- **Drug Deals**
Protease inhibitors can't save your life if you can't afford them.
Out, March 1997
- **Does DHEA Work?**
This wonder hormone is said to boost your sex drive and reverse aging for about 25 bucks a bottle.
Men's Health, March 1997
- **Sleep and Snake Oil**
Melatonin, a powerful, poorly understood hormone faddishly popular as a sleep aid, may in fact be the last thing you should take if you want a restful night.
Discover, March 1997
- **Sleepless in Washington**
Too little sleep can make you sick. But with new medicines and therapies, here's how you can get a good night's rest.
Washingtonian, March 1997
- **Bound and Gagged**
HMOs need to be reformed – but in the right way.
Newsweek, March 17, 1997
- **What You Should Know Whatever Your Age**
Hormones and your health
Family Circle, April 1, 1997
- **Whose right to die?**
America should think again before pressing ahead with the legalization of physician-assisted suicide and voluntary euthanasia.
The Atlantic Monthly, March 1997

Books, Etc.

Book review editor:
Chris Papadopoulos, M.D.

DOC: The Life of Emily Hammond Wilson

Therse Magnotti. Annapolis, MD: Shady Grove Rural Heritage Society, Inc.

144 pages. \$17.95 (hardcover)

D*OC: The Life of Emily Hammond Wilson* is the biography of a female country doctor who practiced for 53 years in lower Anne Arundel County, Maryland, during the early and mid-twentieth century.

The biography is written by a volunteer of the Shady Side Rural Heritage Society. The author, who had never before authored a published book, moved to the Anne Arundel County area after Dr. Wilson had retired. The book is easy reading, and the author has captured the atmosphere of the times while plainly but colorfully describing Dr. Wilson's activities. This volume of only 144 pages is divided into 42 short chapters, each of which focuses on a meaningful event in the doctor's life. (The proceeds of this biography are directed to the Shady Grove Rural Heritage Society.)

The book is written for the general public; however, it is of special inter-

est to physicians. It provides the opportunity to mentally compare the present trials and tribulations of medical practice with those faced by a rural practitioner working before the advent of antibiotics or other modern tools for diagnosis and treatment, frequently delivered via dirt roads, often muddy or covered with ice and snow.

When this biography was written two years ago, Dr. Wilson was still enjoying driving, gardening, entertaining visitors, and attending parties. As a result of a recent fall with residual knee problems, she has had to curtail some of these activities, but still drives despite her almost 93 years. As she is quoted in this book, "I'm going to keep going until I'm old."

MARION FRIEDMAN, M.D.

Dr. Friedman is a retired Baltimore family physician and internist and editor of *The Maryland Family Doctor* and the *Maryland Medical Journal*. ■

NEW HOTLINE FOR ROSACEA SUFFERERS

Rosacea, a chronic and potentially disfiguring facial disorder, is estimated to affect 13 million adults in the United States. This acne-like disease is growing increasingly common as the 76 million Baby Boomers enter their 30s, 40s, and 50s. Among the most famous rosacea sufferers is President Bill Clinton, whose doctors recently disclosed his condition. The National Rosacea Society has a new toll-free hotline, 1-888-NO-BLUSH. Free educational materials can be obtained and the names of area dermatologists who treat this disorder are provided.

1997 Annual Meeting

May 2-3, 1997

Turf Valley Hotel & Country Club

Ellicott City, MD

Maryland's Premier Golf Resort and Conference Center, Turf Valley Hotel and Country Club, is just 20 minutes from Baltimore's Harborplace and 60 minutes from our nation's capital.

Med Chi's annual meeting offers 14 CME Category 1 credits, social gatherings and the newest information in health care delivery options.

Friday CME luncheon - Second Annual Theodore E.

Woodward, MD, Lecture on the History of Medicine

"Milton Winternitz, a Baltimore Boy at New Haven"

Friday MMPAC Luncheon with invited speaker

Congressman Robert Ehrlich

Friday Evening "Meet the Candidates" Reception

Saturday Evening President's Banquet honoring

newly-elected Med Chi president Thomas Allen, MD

Expanded exhibitor offerings give attendees a chance to explore the latest clinical and practice management information and services including: home health and hospice care, pharmaceuticals, rehabilitation and long-term care, HMOs, medical staffing and billing, investments, insurance, banking, telecommunication and computer technologies.

Med Chi has blocked a number of well-appointed sleeping rooms for this meeting. To assure availability, please call Turf Valley 410-465-1500 and tell them you are with Med Chi.

Questions? Call Med Chi's Education and Information Department at 410-539-0872 or 1-800-492-1056.

CME/Educational offerings

Risk Management Issues	Can Managed Care be Managed?
Dermatology	Occupational Asthma
Aging and the Performing Artist	Air Pollution
Lung Reduction Surgery	Elder Abuse
Male Sexuality	Psychiatric Syndromes
Dyspareunia in Women	Ophthalmology for Primary Care
Enhancing Your CME Program	Tobacco: Counseling and
The Internet for Physicians	Pharmaceuticals
Post Acute Services	The Physician and BPQA
Physician's staff programs	

The House of Delegates will meet on Friday morning and Saturday afternoon with Reference Committees on Friday afternoon.



Saturday Keynote Speaker

Steve Wetzell

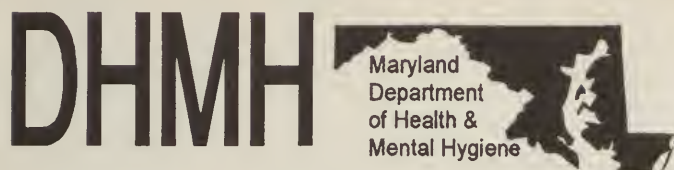
Founder of Minnesota's Buyers Health Care Action Group

On Saturday morning, Med Chi's general session kicks off with the topic **"Can Managed Care be Managed?"** The symposium culminates with a presentation by Steve Wetzell, founder and executive director of Minnesota's Buyers Health Care Action Group (BHCAG). Choice Plus, BHCAG's plan, offers a new model for linking employers and health care practitioners without an insurance middleman. Presently, it provides health care for 400,000 employees, retirees and dependents in Minnesota, Wisconsin, and the Dakotas. First year results generated cost reductions of approximately 11% compared to costs under previously offered plans.

The morning session will present discussions on three fundamental issues associated with the growth of managed care:

- Ethics and capitation—can they co-exist?
- Does managed care advance public health?
- Graduate medical education—future funding?

By presenting an alternative model, Mr. Wetzell's insights and experience offer a provocative conclusion.



EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

April 1997

Changes in the Maryland Immunization Schedule

The Center for Immunization has finalized the new *Maryland Department of Health and Mental Hygiene Recommended Childhood Immunization Schedule*, effective February 3, 1997. These changes have been approved by the Subcommittee on the Immunization and Infectious Diseases of the Medical and Chirurgical Faculty of Maryland. You will note several changes to the attached schedule, as follows:

- 1) DTaP vaccine is now recommended for routine use in infants;
- 2) The "sequential polio schedule", i.e. Inactivated Polio Vaccine (IPV) at 2 and 4 months, with Oral Polio Vaccine (OPV) at 12 months and 4-6 years, is now recommended;
- 3) MMR vaccine is listed at 12 months of age;
- 4) Varicella vaccine is listed at 12 months of age.

DTaP Vaccines Update

Currently *two* acellular pertussis vaccines are licensed for *all five doses* of the diphtheria, tetanus and pertussis vaccination series. On December 30, 1996, Wyeth-Lederle's DTaP, ACEL-IMUNE[®], received FDA licensure for use among infants. **PLEASE NOTE: The previous ACEL-IMUNE[®] formulation is still licensed only for doses 4-5.** Any ACEL-IMUNE[®] currently in your stock should not be used for the first three doses in the series. **The new formulation will have different packaging.** TRIPEDIA[®] (Connaught) DTaP vaccine was the first DTaP (July 1996) to gain approval. The TRIPEDIA[®] formulation has remained consistent and may be used for all doses in the series. Only DTaP vaccines licensed for administration to infants at 2, 4, and 6 months of age and to other children <7 years of age will be offered through the VFC Program.

The schedule is included on the next three pages.

Maryland Department of Health and Mental Hygiene
Recommended Childhood Immunization Schedule
 Approved by the Medical-Chirurgical Faculty of Maryland

Table 1. Primary Immunization for Children Beginning Immunization Under 4 Months of Age

Vaccine	Age	At Birth	2 months	4 months	6 months	12 months	15 months	4-6 years	11-12 years
Hepatitis B		Hep B	Hep B		Hep B				Hep B ⁽¹⁾
Diphtheria, Tetanus, Pertussis			DTaP ^(2,3)	DTaP	DTaP		DTaP ⁽⁴⁾	DTaP ⁽⁵⁾	Td ⁽⁶⁾
Haemophilus influenzae type b			Hib	Hib	Hib ⁽⁷⁾		Hib ⁽⁸⁾		
Polio			IPV ⁽⁹⁾	IPV ⁽⁹⁾		OPV ⁽⁹⁾		OPV ⁽⁹⁾	
Measles, Mumps, Rubella						MMR ⁽¹⁰⁾		MMR ⁽¹¹⁾	MMR ⁽¹¹⁾
Varicella						Var ^(10,12)			Var ⁽¹²⁾

March, 1997

Table 2.

**PRIMARY IMMUNIZATION FOR CHILDREN BEGINNING
IMMUNIZATION BETWEEN FOUR (4) MONTHS AND
SIX (6) YEARS OF AGE**

First Visit (≥ 4 mos. of age)	DTaP, Polio (9), Hib, Hep B. Varicella and MMR should be given as soon as child is age 12 months.
Second Visit (1 mo. after first visit)	DTaP, Hib (7), Hep B
Third Visit (1 mo. after second visit)	DTaP, Polio, Hib (7)
Fourth Visit (6 wks. after third visit)	Polio
Fifth Visit (≥ 6 mos. after third visit)	DTaP, Hib (7), Hep B
Additional Visits Age 4-6 yrs	DTaP (5), Polio, MMR (preferably at or before school entry)
Age 11-16 yrs	Td- Repeat every 10 yrs throughout life

Table 3.

**IMMUNIZATION SCHEDULE FOR PERSONS
SEVEN YEARS OF AGE AND OLDER WHO WERE
NOT VACCINATED AT THE RECOMMENDED
TIME IN EARLY INFANCY**

First Visit	Td, Polio(13), MMR,HepB, Varicella (12)
Second Visit (6-8 wks. after first visit)	Td, Polio, MMR, Hep B. Varicella (give 2 doses of Varicella if person was ≥ 13 years old at first dose)
Third Visit (6 mos. after second visit)	Td, Polio, Hep B
Additional Visits (≥ 5 yrs after third visit)	Td - Repeat every 10 years throughout life.

**INFANTS AND CHILDREN SHOULD ALWAYS
RECEIVE ALL VACCINES WHICH ARE
INDICATED AT EACH VISIT
(i.e., simultaneous administration).**

See next page for notes

Refer to statements from the American Academy of Pediatrics Red Book Committee, the Advisory Committee on Immunization Practices of the U.S. Public Health Service, and package inserts for each vaccine for additional details.

1. Hep B - Hepatitis B vaccine. Children who have not previously received 3 doses of hepatitis B vaccine should initiate or complete the series during the "5th grade year." The 2nd dose should be administered at least 1 month after the 1st dose, and the 3rd dose should be administered at least 4 months after the 1st dose and at least 2 months after the 2nd dose.
2. DTaP - Diphtheria and tetanus toxoids combined with acellular pertussis vaccine for children less than seven (7) years of age. Use DT pediatric vaccine when pertussis vaccine is contraindicated.
3. DTP - Diphtheria and tetanus toxoids combined with whole cell pertussis vaccine is also licensed for children less than seven (7) years of age. Use DT pediatric vaccine when pertussis vaccine is contraindicated. DTP/Hib combined vaccine is available.
4. The 4th dose of DTaP or DTP can be given as early as 12 months of age if given at least 6 months after the 3rd dose of DTaP or DTP.
5. If the 4th DTaP or DTP is administered after the 4th birthday, a 5th DTaP or DTP is not necessary.
6. Td (adult) - Tetanus and diphtheria toxoids for children 7 years of age and older, and for adults.
7. Six month dose of Hib may not be needed depending on the brand of Hib conjugate vaccine; check manufacturer's instructions. Four doses may not be needed if series begun late in infancy; one dose at ≥ 15 months of age precludes the need for more doses.

Always follow the manufacturer's recommendations regarding dosage, route of administration, and storage of vaccines.

8. Hib - Haemophilus influenzae type b conjugate vaccine is recommended for children only up to age 5. DTP/Hib combined vaccines are available.
9. IPV refers to enhanced inactivated polio vaccine and OPV refers to live oral polio vaccine. OPV may be given at 2, 4, 6 months and 4-6 years; IPV may be given at 2, 4, and 15 months and 4-6 years. The preferred schedule is IPV at 2, 4 months and OPV at 12 months and 4-6 years.
10. MMR vaccine and varicella vaccine should be administered on/after the first birthday.
11. The 2nd dose of MMR is routinely recommended at 4-6 years of age (school entry), or at 11-12 years of age if not given earlier. It may be administered at any visit ≥ 12 months of age, provided at least 1 month has elapsed since receipt of the 1st dose.
12. Varicella zoster virus (chickenpox) vaccine can be administered to susceptible children any time at or after 12 months of age. Unvaccinated children who lack a reliable history of chickenpox disease or prior varicella vaccination should be vaccinated at the 11-12 year old visit. Susceptible persons ≥ 13 years of age should receive two (2) doses, 4-8 weeks apart.
13. Persons seven years of age and older should ask their doctor about the preferred polio vaccine.

Routes of vaccine administration

Intramuscular - Hep B, DTaP, DTP, Td, DT, Hib
Subcutaneous - MMR, IPV, Varicella
Oral - OPV

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

MGA\MGS series, Department of Mental Hygiene, The Johns Hopkins University, School of Hygiene and Public Health, Keswick Nursing Center, 40th Street, 6:00 p.m. -7:00 p.m., light refreshments served at 5:30 p.m. Credits: TBA. Sponsored by the Maryland Chapter of the American Geriatrics Society (AGS) and the Maryland Gerontological Association (MGA), with assistance of the Geriatrics Committee of the Maryland Academy of Family Physicians. Info: Donna Meisel Weinreich, 410-675-3244 (e-mail: dmeisel@umabnet.ab.umd.edu) or Joseph J. Gallo, M.D., M.P.H., 410-955-0599 (e-mail: jgallo@welchlink.wlech.jhu.edu).

Pain management in the older adult

Apr. 29

25th annual pediatric trends, Johns Hopkins Medical Institute, Department of Pediatrics. This course provides a comprehensive update on new developments of interest to practitioners who care for infants, children, and adolescents. Credits: 42.5 Cat 1 AMA credits; 45.5 AAP credits; 37.5 AAFP prescribed hours.

Apr. 14-19

38th annual postgraduate institute for pathologists in clinical cytopathology, sponsored by The Johns Hopkins University School of Medicine. Credits: 94.5 Cat 1 AMA credits plus up to 10 hrs. video instruction. Fee: \$2450/physicians; \$1300/senior residents.

Course A (Home study)

Apr.

Course B, concentrated lecture and laboratory studies, Johns Hopkins Medical Institutions, Baltimore, MD.

Apr. 14-25

Seventh annual clinical care of the patient with HIV infection, sponsored by the Department of Medicine, at the Renaissance Harborplace Hotel, Baltimore, Maryland. Credits: 15 Cat 1 AMA credits. Fee: \$375/physicians; \$190/residents, fellows, allied health professionals.

Apr. 17-18

11th annual mood disorders symposium, sponsored by The Johns Hopkins Affective Disorders Clinic, and DRADA. Credit: Cat 1 AMA credit; Cat A credit; Md. State Board of Examiners of Psychologists; Md. State Board of Examiners for Social Workers. Fee: \$50/DRADA members; \$60/other attendees.

Apr. 30

Institute on ministry with the sick, sponsored by the Johns Hopkins University School of Medicine. Credits: 14 Cat 1 AMA credits. Fee: \$150.

May 5-7

Pediatric allergy and immunology for the practitioner, sponsored by the Division of Pediatric Allergy and Immunology. Credits: up to 14 Cat 1 AMA credits. Fee: If postmarked by April 1, \$275/physicians; \$200/residents, other allied health professionals. If postmarked after April 1, \$295/physicians; \$220/residents, other allied health professionals.

May 8-9

Lipid disorders training program - advanced update, sponsored by the Johns Hopkins University School of Medicine and the Johns Hopkins Lipid Clinic at the Renaissance Harborplace Hotel, Baltimore. Appropriate credit is pending.

May 9

Critical issues in surgical pathology, sponsored by the Department of Pathology. Credits: 14 Cat 1 AMA credits. Fee: \$400/physicians; \$200/residents, fellows, students.

May 9-10

42nd annual topics in clinical medicine, sponsored by the Department of Medicine. Credits: 39 Cat 1 AMA credits. Fee: \$750/physicians; \$600/residents, fellows, other professionals.

May 12-16

Lipid disorders training program - basic course, sponsored by the Johns Hopkins University School of Medicine and the Johns Hopkins Lipid Clinic at the Renaissance Harborplace Hotel, Baltimore. Appropriate credit is pending.

May 28-30

Fifth annual advanced topics in CT with emphasis on spiral CT, sponsored by the Department of Radiology, at the Eldorado Hotel, Sante Fe, New Mexico. Credits: 16 Cat 1 AMA credits. Fee: \$525/physicians; \$475/residents, fellows.

July 24-27

The Johns Hopkins Medical Institutions (continued)

Continuously throughout the year

Visiting preceptorship in pediatric critical care medicine. Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.

The department of radiology and radiological sciences offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.

Visiting physicians. Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.

Johns Hopkins medical grand rounds. Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.

Johns Hopkins sports medicine grand rounds. Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Self-Directed CME Activities

Disease management of lipid disorders (audio tape and test). Credit: 1 Cat 2 AMA credit. Expires 6/97.

CD-ROM-based interactive multimedia radiology teaching file for Mac or PC w/single user licenses (SUL), site licenses (SL), or multisite licenses (MSL). Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Office of Continuing Medical Education, Washington University School of Medicine, Campus Box 8063, 660 South Euclid Ave., St. Louis, MO 63110-1093. Unless otherwise noted, seminars will be held at the Washington University Medical Center, Eric P. Newman Education Center (EPNEC), 320 S. Euclid Ave., St. Louis, MO 63110. Info: Cathy Sweeney, 800-325-9862, Fax 314-362-1087.

Internal medicine review, Monday evenings, The Jewish Hospital.

Apr.–May

Pitfalls & treatment of anxiety plus depression, 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598.

Apr. 16

Sixth annual meeting and clinical congress of the American Association of Clinical Endocrinologists (AACE), Marriott, Philadelphia, Pennsylvania. Credits: up to 36.5 Cat 1 AMA credits. Info: 904-353-7878.

Apr. 16–20

Problem solving in imaging of the brain, spine, and head and neck, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education at The Ritz-Carlton Resort Hotel, Amelia Island,

Apr. 17–20

Miscellaneous (continued)

Florida. There will be both didactic lectures and workshops. Credits: 26 Cat 1 AMA credits. Fee: \$650/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

Expanding the cardiac continuum, presented by the Heart Institute at St. Joseph Medical Center at the Hunt Valley Marriott. CME credits available. Fee: \$100/physicians; \$80/nurses, other healthcare professionals. Info: 410-337-1309. **Apr. 18**

2nd annual angio/interventional review course, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Credits: 10 Cat 1 AMA credits. Fee: \$215/physicians; \$155/residents, fellows, full-time military, U of F radiology alumni (\$135 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 19-20**

9th annual radiology review course: "what you need to know," sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Attendees will improve their knowledge of differential diagnosis, imaging patterns, and techniques of examination. Credits: 50 Cat 1 AMA credits. Fee: \$695/physicians; \$525/residents, fellows, full-time military, U of F radiology alumni (\$475 each for two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 20-25**

Cytochrome P450 enzyme system, 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598. **Apr. 23**

Adult immunization: strategies that work, presented via satellite by the Centers for Disease Control and hosted by the Maryland Department of Health & Mental Hygiene. Fee: none. Info: Sandra Kash, 410-767-6679. **Apr. 24**

Critical care medicine '97: 11th annual review and update, sponsored by the Center for Bio-Medical Communication, Inc., Hyatt Regency, Washington, D.C. Credits: 41.25 Cat I AMA credits, 41.25 AAFP. Fee: By Mar. 21, 1997, \$795/physicians; \$575/physicians-in-training, allied professionals. Info: 201-385-8080, Fax 201-385-5640 (e-mail: cbcbiomed@aol.com). **Apr. 30-May 4**

2nd annual mammography review course, sponsored by The University of Florida, College of Medicine, at The Buena Vista Palace, Orlando, Florida. Course is designed as an overview of the practical aspects of breast imaging, including interventional procedures. Credits: 15 Cat I AMA credits. Fee: \$295/physicians; \$215/residents, fellows, full-time military, U of F Radiology Alumni (\$185 each, two or more residents registering at the same time from the same institution). Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Apr. 25-27**

Cancer prevention in community practice, sponsored by the Medical and Chirurgical Faculty of Maryland, at Shady Grove Adventist Hospital, Montgomery County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **May 2**

Fourth annual osteoporosis and other metabolic bone diseases symposium, Doubletree Inn at the Colonnade. Credits: 9.5 Cat 1 AMA credits; nursing and PT CEUs pending. Fee: \$100/physicians; \$90/fellows, nurses, and physical therapists. Info: Sherry Buchman, 410-554-2923, Fax 410-554-6794. **May 2-3**

Clinical auscultation of the heart, Georgetown University Medical Center, Washington, D.C. Sponsored by the American College of Cardiology. Credits: 21 Cat 1 AMA credits. Contact: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, MD 20814-1699, 800-253-4636, ext. 695, Fax 301-897-9745. **May 7-9**

Miscellaneous (continued)

56th annual American occupational health conference: discover the reality , sponsored by the American College of Occupational and Environmental Medicine (ACOEM) in conjunction with American Occupational Health Conference (AOHC). Orange County Convention Center, Orlando, Florida. 39 concurrent scientific sessions, 42 postgraduate seminars, and 7 two-day training courses. Contact: Kay Cone, ACOEM, 55 W. Seegers Rd., Arlington Heights, IL 60005, 847-228-6850, ext. 152, Fax 847-228-1856.	May 9–16
Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Physician Memorial Hospital at Hamilton Center, Charles County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.	May 13
Gay and lesbian issues in psychiatry and psychotherapy , 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598.	May 14
TraumaCare '97: the 10th annual trauma anesthesia and critical care symposium and world exposition , Baltimore, MD. Info: ITACCS, P.O. Box 4826, Baltimore, MD 21211, Fax 410-235-8084.	May 15–17
Cutaneous melanoma '97: a clinical symposium for primary care practitioners , sponsored by The Skin Cancer Foundation and Memorial Sloan-Kettering Cancer Center, Memorial Sloan-Kettering Cancer Center, New York, NY. Credits: 6.5 Cat 1 AMA credits. Info: Ludmilla Popoff, 212-639-6754.	May 16
2nd Annual mammography — practical challenges of the '90's , sponsored by X-Ray Associates of New Mexico, P.C., at The Eldorado Hotel, Santa Fe, New Mexico. Credits: 20 Cat 1 AMA credits. Fee: \$650/physicians; \$450/residents, fellows; \$350/technologists, nurses. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	May 23–26
How to prevent successful lawsuits in mental health , 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598.	May 28
Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Fallston General Hospital, Harford County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.	June 4
Epidemiology & prevention of vaccine-preventable diseases , a four-part comprehensive course, presented via satellite by the Centers for Disease Control and hosted by the Maryland Department of Health & Mental Hygiene. Fee: none. Info: Sandra Kash, 410-767-6679.	June 5, 12, 19, 26
Family interventions in severe mental illness , 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598.	June 11
Uroradiology in Santa Fe '97 , sponsored by the American College of Radiology and the Society of Uroradiology, at the Eldorado Hotel, Santa Fe, New Mexico. Credits: Cat 1 AMA credits TBA. Fee: \$595/physicians; \$395/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	June 15–18
Imaging in Santa Fe , sponsored by the American Association of Physician Specialists and the International Institute for Continuing Medical Education, at the Eldorado Hotel, Santa Fe, New Mexico. Credits: 25 Cat 1 AMA credits pending . Fee: \$625/physicians; \$425/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	July 28–Aug. 1

Miscellaneous (continued)

Self-Directed CME Activities

Maryland physicians' campaign against family violence, module one: domestic violence, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Maryland physicians' campaign against family violence, module two: child maltreatment, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

Fluorescein angiography conference, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00 a.m. – 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.

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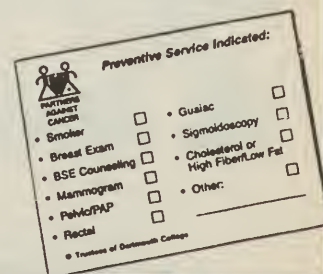
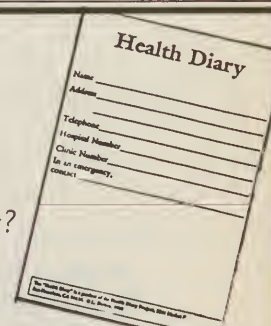
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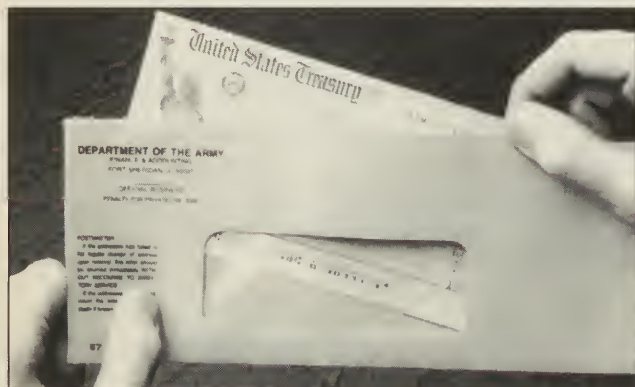


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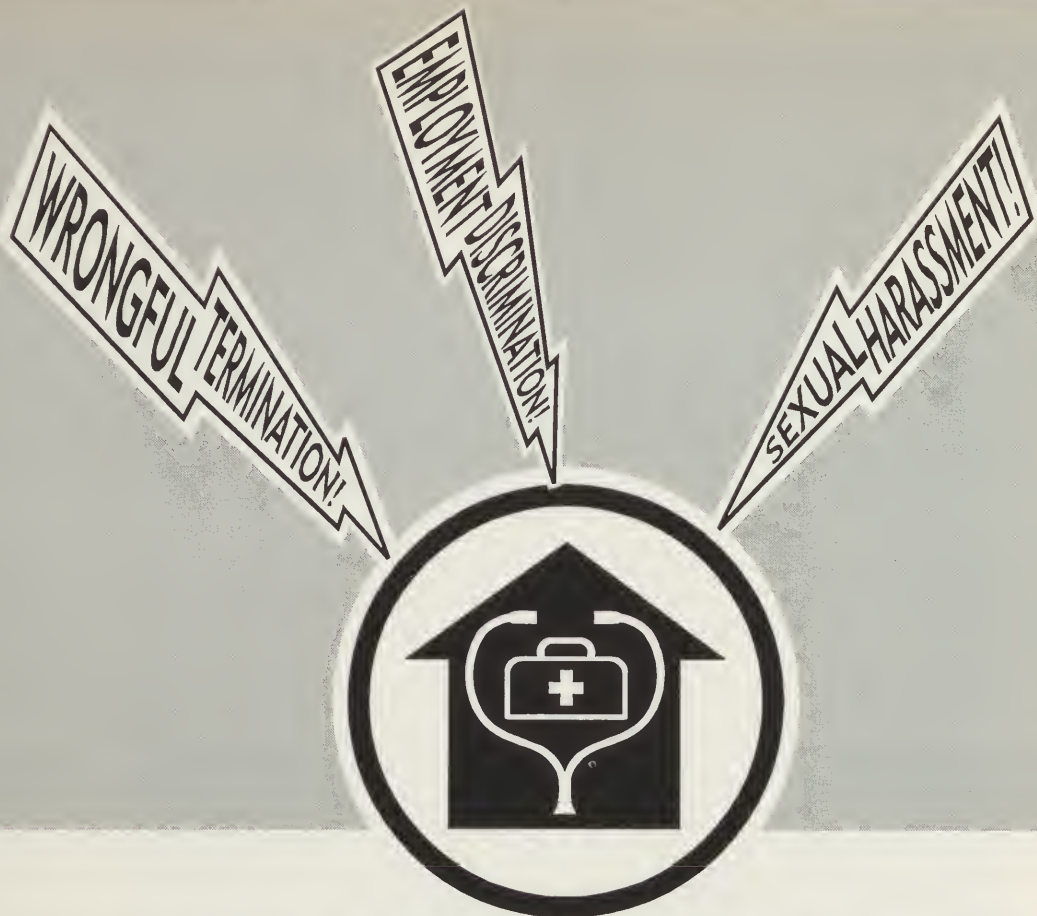
▼ As many as 50% of patients with coronary heart disease do not follow risk reduction therapies for a full year. Randomized clinical trials have demonstrated that risk reduction therapies, including smoking cessation, lipid lowering, exercise, aspirin therapy, anticoagulant therapy, ACE inhibitor therapy, beta-blocker therapy, and blood pressure control, can be of significant benefit to patients with cardiovascular disease. American Heart Association recommendations for implementation of risk reduction case management can extend survival, improve quality of life, and decrease the need for procedures such as bypass grafting and angioplasty (*Am Fam Phys* 1997;55(2): 491-500).

▼ A study designed to examine the role of anxiety and depression symptoms in the development of hypertension found that an association does exist between self-perceived presence of anxiety or depression symptoms and subsequent onset of hypertension (*Arch Fam Med* 1997;6:43-49). The study included 2 992 men and women who initially showed no signs of hypertension. The study participants were followed for 7 to 16 years. The authors suggest that the findings of this study indicate the reduction of anxiety and depression is important for all patients, not just those with clinical implications.

▼ A University of Michigan study of hospitalized psychiatric patients discovered a relationship between the diagnosis of borderline personality and the possession of transitional objects as adults. A sizable percentage of persons with the diagnosis either brought their teddy bear, blanket, or special object to the hospital or acknowledged using one at home. While the phenomenon of transitional objects is thought to provide security to many children, the role for adults is not well understood. Perhaps the unstable emotions and sense of emptiness associated with the personality disorder are settled by the close inanimate attachment (*Am J Psychiatry* 1997;154: 250-255).

▼ Superior language skills in females may be due to anatomical differences. Results of a study published in the *Archives of Neurology* (1997;54:171-176) show that the Wernicke language area and the Broca language area are larger in females than in males. The authors conclude that these anatomical differences may account for females tendency to excel at verbal skills tests, while males have a tendency to excel at mathematically based tests.

▼ According to the findings of a study analyzing those with childhood-onset obsessive-compulsive disorder and chronic tic syndrome, a genetic marker for these diseases may exist (*Am J Psychiatry* 1997; 154:402-407). B Lymphocyte Antigen D8/17, thought to be a marker for susceptibility to rheumatic fever, was expressed in the subjects (who had no history of rheumatic fever). The authors concluded that the presence of D8/17 indicated obsessive-compulsive disorder or Tourette's syndrome may be alternate expressions of a susceptibility to rheumatic fever.



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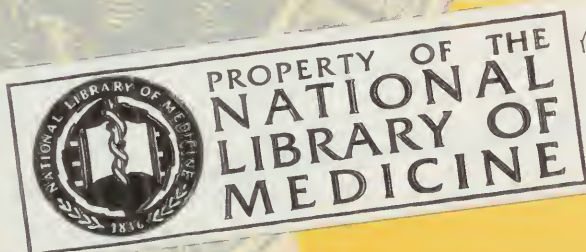
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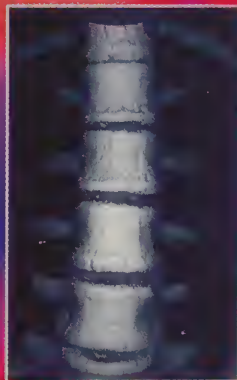
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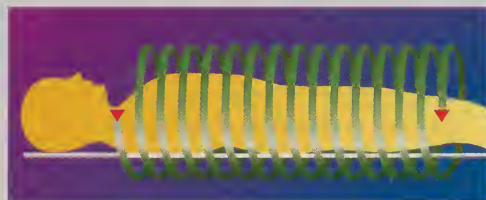
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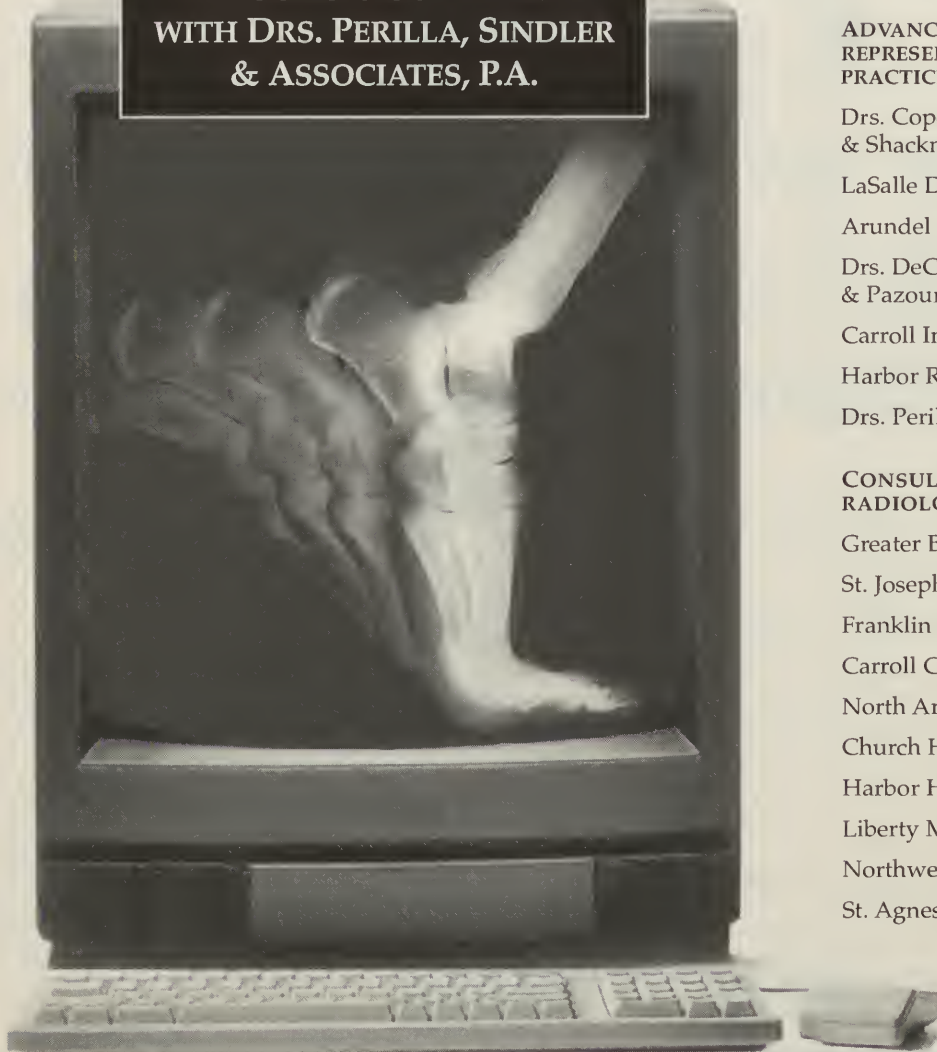
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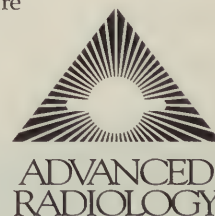
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Editor's Introduction

We are indebted for the production of this special issue on skin cancer and related subjects to David Freitag, M.D., a dermatologist from Bethesda who practices Mohs micrographic surgery and is a member of our editorial board. In developing this issue, Dr. Freitag has been motivated, not by obtaining original works or unusual cases which would be primarily of interest to other dermatologists, but instead, to present the more common and practical aspects of the subject which might not be well known to other types of practitioners. It is hoped that after reading these presentations, when patients are seen for other reasons, the physician may be more prone to consider the etiology of the incidentally noted lesion.

We are also especially grateful to two pharmaceutical companies who contributed to our efforts to disseminate the message of care in the sun. Without the educational contributions of Schering Oncology/Biotech and Roche Pharmaceuticals, this issue would not have been possible. The Skin Cancer Foundation (1-800-SKIN-490) also made possible the bookmark inserts to promote the early diagnosis of melanoma.



MARION FRIEDMAN, M.D.

Guest Editor's Introduction

Anually, the month of May is dedicated as "National Skin Cancer Awareness Month" by the American Academy of Dermatology. I am grateful to the Maryland Medical Journal for supporting these efforts with the publication of this issue. Herein we offer seven articles contributed by expert Maryland physicians centered around the current epidemic of cutaneous malignancies. The overwhelming majority of such tumors are caused by excess exposure to ultraviolet radiation.

Warwick Morison, M.D., (Towson) describes sunlight as an environmental toxin. His article is a useful reminder for physicians and patients that "sun sense" is in order as we begin another season of outdoor activity. Matthew Katz, M.D., (Rockville) reviews basal cell and squamous cell carcinomas, the most common nonmelanoma skin cancers, whose increasing numbers have become a major societal concern. John Skouge, M.D., (Lutherville) reviews Mohs micrographic surgery for skin cancers. This technique offers precise margin control and significantly improved cure rates for difficult, unusual, or recurrent skin cancers. Dr. Skouge is president-elect of the American College of Mohs Micrographic Surgery and Cutaneous Oncology.

The epidemiology of melanoma is also raising concern nationwide. Its incidence is accelerating more rapidly than any other cancer. Jean Henneberry, M.D., and Susan Koch, M.D., (Baltimore) discuss and illustrate a common precursor to melanoma, the atypical mole syndrome. Gary Peck, M.D., (Kensington) describes a relatively new technique useful in the early diagnosis of pigmented lesions, epiluminescence microscopy. Julie Lange, M.D., (Baltimore) updates the surgical management of invasive primary melanoma, including recent developments in sentinel node biopsy techniques and adjuvant immunochemotherapy for advanced melanoma. Lastly, Allan Harrington, M.D., (Hunt Valley) and I characterize a number of uncommon cutaneous malignancies which may need consideration in one's differential diagnosis if they are to be diagnosed and treated early.

It is my hope that the contributing authors have provided some understanding to the physicians of Maryland about the growing dilemma of ultraviolet damage and subsequent cutaneous neoplasia.

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Sunlight: An environmental toxin for humans

A primer to advise patients

Warwick L. Morison, M.D.

Dr. Morison is professor of dermatology, The Johns Hopkins University, Baltimore, and maintains a private practice in Towson.

ABSTRACT: *Sunlight causes acute toxic effects such as sunburn, local and systemic immune suppression, and long-term adverse effects including photoaging and skin cancer. The degree of damage depends on the overall exposure-dose and individual susceptibility. Various strategies should be employed to minimize sun-exposure damage, including proper use of sunscreens and exposure avoidance. Public education might best be focused on protecting children and promoting awareness of photoaging changes in adults.*

Sunlight is essential to life, but for the skin and other organs, it is an environmental toxin. The amount of damage sunlight causes depends on the type and amount of exposure and the susceptibility of the individual to its toxic effects.

The various wavelengths of sunlight radiation are divided according to how it effects people (**Figure 1**). The ultraviolet (UV) portion of the spectrum contains the most energetic photons and is responsible for most of the sun's biologic effects. In Maryland, sunlight contains little radiation shorter than 300 nm, but a sliver of UVB (300 nm to 315 nm) radiation causes 80% to 90% of such toxic effects as sunburn, photoaging, and skin cancer. UVB radiation is the most variable portion of sunlight because it is scattered and absorbed by ozone and water vapor in the atmosphere. When it has a long passage through the atmosphere, most UVB radiation is removed from sunlight. Therefore, there is little UVB radiation in sunlight during winter, or at 9:00 a.m. or 6:00 p.m. in summer. Air pollution is another major determinant of the UVB content of sunlight. Despite the decreasing ozone layer, the UVB reaching the earth has decreased due to pollution. UVA (315 nm to 400 nm) radiation is

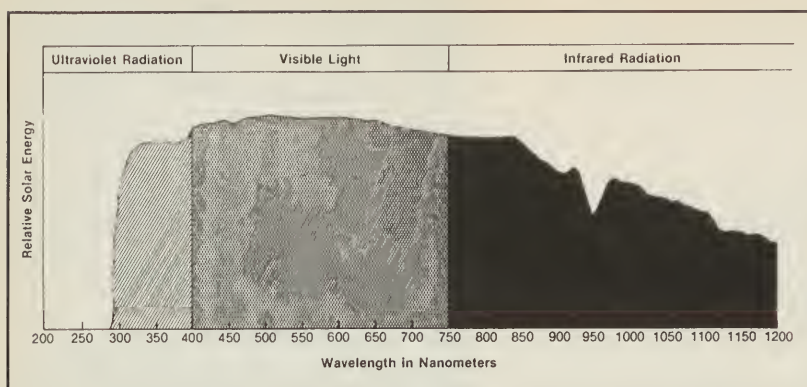


Figure 1. Emission spectrum of sun at surface of earth.

much more abundant in sunlight, but it causes a smaller proportion of damage to the skin. Similarly, visible light, while being essential for vision, does not produce much photochemical change in skin. Infrared radiation has only thermal effects.

The skin's susceptibility to sunlight varies greatly and, in an attempt to classify this variation, a system of skin "typing" has crept into use (Table 1). Caucasians are classified into skin type I to IV according to their response to a reasonable dose of sunlight, such as a 30- to 60-minute exposure around noontime at the beginning of summer. People with darker skin are classified by skin color into skin types V and VI. The system is imperfect because there is much overlap, particularly between skin types II and III and between IV and V. Regardless, the system is useful because sunlight is much more toxic for a person with skin type I than skin type III, and for skin type II than skin type IV. Other phenotypic traits, such as hair color and eye color, give some indication of susceptibility. For example, a blue-eyed, blonde-haired person of skin type II is more susceptible to skin cancer than a brown-eyed, dark-haired person of the same skin type.

This review is not intended to be all-inclusive, but instead, to highlight a few of the harmful effects of sunlight and provide some practical comments. There are some very comprehensive books available on photomedicine to provide more detail.^{1,2}

Acute effects of sunlight

Sunburn. By far the most common adverse effect of sunlight exposure, and usually short-lived and self-healing, sunburn is mainly due to UVB radiation with contribution of up to 20% from UVA radiation. Skiing at 10,000 feet and visiting a UVA suntan parlor are two less common situations when UVA radiation will be the main cause of a sunburn. There is evidence sunburns in

childhood are more harmful in terms of subsequent development of melanoma and nonmelanoma skin cancer as compared to sunburns in adult life, perhaps because they are more severe and frequent. Sunburn is classified according to its color, pink through bright red, and the presence or absence of blistering. The use of the classification of thermal burns (i.e., first, second, and third degree) for sunburns is very misleading because sunlight-induced burns do not scar. A severe sunburn can cause a loss of melanocytes, and therefore hypopigmentation, but this is not associated with scarring.

Finally, topical and oral corticosteroids have no effect on the course of a sunburn, except perhaps making the patient feel happier if given in a high dose.

Tanning. Delayed pigmentation of the skin following exposure to sunlight is a defensive and reparative response and not a harmful effect. However, it is evidence the skin has been injured. A tan is mainly due to UVB radiation, but under certain circumstances, for example, while skiing in winter, a tan may be mainly due to UVA radiation; such a tan is a different color with an orange hue. It is important to remember that a tan is even brown pigmentation and not just an eruption of freckles. Many redheads will claim they tan, but they are referring to the extreme damage their skin has suffered and the subsequent appearance of freckle masses.

Immunologic Effects. Exposure to sunlight produces marked alterations in immunity. Photoimmunology, the research field devoted to study of these changes, has mainly concentrated on experimental animals, but when examined, similar changes have been found in people. T-cell responses, particularly delayed-type hypersensitivity such as contact allergy, are suppressed, and this suppression is systemic,

Table 1. Skin types I-IV determined by response to first exposure to sunlight for summer.
Skin types V and VI based on skin color.

Skin type	History	Examination
I	Always burn, never tan	
II	Always burn, sometimes tan	
III	Sometimes burn, always tan	
IV	Never burn, always tan	
V		Brown _a
VI		Black

_a Chinese, Mexican, American Indian.

lasting days or weeks. The teleologic explanation for this suppression may be that it protects from development of autoimmune reactions to the large number of abnormal photoproducts released from sun-damaged skin. Sun-induced alterations in immunity may play a role in the development of skin cancer and the response to infectious agents, particularly tropical diseases.

Chronic effects of sunlight

Photoaging. Photoaging is different from chronologic aging of the skin; any person who doubts this should compare the skin on their buttocks to that on their face. The main changes of photoaging are freckling, lentigos, hypopigmented areas, telangiectasia, bruising, wrinkling, and actinic keratoses. It is mainly due to UVB radiation, but UVA radiation makes a significant contribution; visible light and infrared radiation also contribute. Studies in animals have established it to be dose-dependent for a given type of skin. All skin types are affected by photoaging, but in people with dark skin the changes develop later and are somewhat different. A yellowish complexion with deeper, thicker wrinkles occurs in these skin types; this may reflect deeper damage in the dermis rather than in the epidermis.

Skin cancer. Well over a million cases of skin cancer occur each year in the United States, and in sun-drenched countries with a fair-skinned population such as Australia, skin cancer has become a major public health problem. The incidence rates are climbing each year due to an aging population, increased time for leisure, focus on outdoor activities, and a progressive decline in the surface area of clothing. Nonmelanoma skin cancer accounts for the most skin cancers, but the incidence of melanoma has been rising at a much faster rate over the last four decades. It is becoming a major cause of cancer mortality.

Sunlight exposure is the major factor in the development of skin cancer, but the relationship between such exposure and the different types of skin cancer varies. Squamous cell carcinomas are mostly dose-related and occur most frequently on maximally-exposed areas of the head, neck, and hands. Basal cell carcinomas, by far the most frequent type of skin cancer, are less directly related to the exposure dose. These lesions occur on exposed skin but are often found in relatively protected sites around the nose and eyes. The relationship between exposure to sunlight and the development of melanoma is the most obscure and the subject of much continued debate. Summarily, everybody agrees sunlight is a major factor in causing melanoma, but another unknown factor or combination of factors also plays a significant role.

UVB radiation plays a dominant role in the development of nonmelanoma skin cancer, but based on animal studies, UVA radiation also contributes. There is evidence visible light could be involved in causing melanoma.

Prevention

Various strategies have been suggested and developed for preventing the harmful effects of exposure to sunlight. All interfere in some measure with enjoyment of outdoor activities, so they must be coupled with a large dose of public education to be successful.

Avoidance of sunlight. People who are very sensitive to sunlight, such as those with skin type I, frequently learn by themselves to avoid beach vacations, choose hobbies other than boating and fishing, and to use the middle of the day for museums rather than strolling in a park. Sunlight in summer before 10:00 a.m. and after 4:00 p.m. is fairly nontoxic because the amount of UVB radiation is low.

Clothing. A hat is an excellent form of protection, particularly for the bald-headed man. A hat with a wide-brim provides protection for much of the face and ears, areas that are prone to damage. Any clothing opaque to visible light will also be opaque to UV radiation. Transmission is increased when clothing is wet, and this is perhaps most obvious in people wearing a white bathing suit. Several companies market lines of clothing designed to protect against exposure to sunlight.

Sunscreens. True sunscreens first became available in the 1970s and have now replaced the old tanning oils, which offered little protection. They are now offered in a bewildering array in stores, but the choice and use of a sunscreen can be guided by a few considerations:

► Sun Protection Factor

Sun Protection Factor (SPF) is a measure of the effectiveness of a sunscreen. An SPF 15 sunscreen, if applied in the same manner as in laboratory tests (2μl/cm²), will provide protection against exposure to 15 minimal sunburning doses. This dose is calculated to be the maximal exposure received by a fair-skinned person in the continental United States during a day. Any person who needs to use a sunscreen should buy one with SPF 15 or higher.

► Classification

Sunscreens are divided into chemical and physical protectants according to their ingredients. Chemical sunscreens usually contain mixtures of cinnamates, benzophenones, and sometimes para-aminobenzoic acid

(PABA), and they act by absorbing UVB and, to some extent, UVA radiation. This summer, many sunscreens will also contain avobenzone, which is a more effective screen for UVA radiation. Physical sunscreens, often called "nonchemical sunscreens," a classic non sequitur, contain chemicals which reflect and scatter UV and visible radiation; titanium dioxide is the main ingredient. Most people prefer chemical sunscreens because they are invisible on the skin.

• **Substantivity**

Substantivity is a measure of how long a sunscreen will adhere to the skin. The choices are non-waterproof, water-resistant, or waterproof. Clearly, the choice should always be waterproof, since most outdoor activities involve either perspiring or water contact.

• **Use**

Sunscreens should be applied 15 minutes before exposure to allow adhesion to the skin and should be applied again after a couple of hours. Several studies have shown that under ideal circumstances most people apply only 50% of the required amount. A good rule-of-thumb is that a four ounce bottle of sunscreen contains enough to cover the exposed area of a 70Kg male in a bathing suit only four times.

• **Adverse reactions**

Most sunscreen ingredients are contact irritants and when applied in high concentration, particularly on the face, will cause stinging and burning. True contact allergic reactions to sunscreens are extremely rare.

• **Which sunscreen should your patients buy?**

You should advise your patients to choose a base that is most pleasant for their skin, select an SPF 15 or higher, and make sure it is waterproof. Also advise them to avoid spending money on fancy brands, packages, and perfumes. All sunscreens have to meet FDA standards, so the discount brand will be as good as the one at the cosmetic counter. Of course, they must be told to use it liberally.

Systemic protection

It would be much more convenient to have a pill available to provide systemic protection from the harmful effects of sunlight. Suffice it to say, many have been tried and all have failed. There is no known systemic agent that provides significant protection from sunlight.

Public education

An important component of any program aimed at protecting people from sunlight exposure is to educate about the dangers as well as the means of protection. Much could be said about such education, but two points are often overlooked. First, the most unprotected segment of the population is children. It has been estimated that 75% of all nonmelanoma skin cancer could be prevented by adequate use of sunscreens before the age of 20 years. Second, attempting to sensitize people to the risk of skin cancer as a means of changing sun-exposure habits is not a very effective approach. Warning about the risk of photoaging is a far better strategy. People can observe photoaging on their own skin and on the skin of people around them, so it is easy to assess the peril ahead. In contrast, few people really understand what a skin cancer looks like or what it means to their health and welfare.

Natural photoprotection

An increase in melanin and thickening of the epidermis provides protection against subsequent exposure to sunlight. A question frequently raised is the value of prophylactic tanning before exposure to sunlight either by incremental exposure to sun or use of tanning devices. The answer is unknown. It is likely the risk/benefit ratio is poor for a skin type II subject and moderate for a skin type IV subject.

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Mohs micrographic surgery for the treatment of difficult skin cancers

John W. Skouge, M.D.

Dr. Skouge is in private practice in Baltimore and is assistant professor of dermatology, The Johns Hopkins University, Baltimore.

ABSTRACT: *Most skin cancers can be managed effectively using standard therapeutic methods. However, specific subsets of skin cancers—including tumors that are recurrent, large, or aggressive, along with tumors located at sites of functional and cosmetic concern—provide a significant therapeutic challenge. Mohs micrographic surgery offers the greatest potential for cure of difficult tumors, while providing for maximal preservation of healthy tissue. This paper describes the history of Mohs surgery, the technique, and the indications for its use.*

Introduction

The incidence of skin cancer is increasing at an alarming rate. The American Cancer Society¹ estimates that there are over 800 000 new non-melanoma skin cancers per year in the United States. Most of these cancers are represented by basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). While these cancers are characterized by low risk for metastasis and death, they carry the potential for local destruction and significant morbidity.

In the present managed care environment, primary care physicians are increasingly expected to manage skin cancer, which in the past, was referred to specialists. Certainly, many primary skin cancers can be successfully managed by the family practice physician, internist, or dermatologist. There exists, however, a subset of cancers that, in a predictable manner, will be poorly responsive to standard treatments and must be considered for alternative therapeutic intervention.

Mohs micrographic surgery, an office-based procedure performed under local anesthesia, is ideally suited to treat these more aggressive



Figure 1. This figure demonstrates subclinical tumor extensions and eccentric pattern of growth.

cancers. Since there are no additional operating room, anesthesia, or pathology costs associated Mohs surgery, it is a cost-effective therapeutic option.

This paper describes the history of Mohs micrographic surgery, the technique, and indications for referral, and differentiates between Mohs surgery and standard frozen section margin control in the operating room.

History

In the 1930s, while still in medical school, Frederick Mohs²⁻⁴ developed a surgical approach for the treatment of those neglected and multiple recurrent skin cancers for which there were no good alternative techniques. At the time, medical opinion held that it was dangerous to cut into a skin cancer for fear that it would cause the cancer to spread. Therefore, Mohs developed a chemical fixative paste that, when applied to the surface of a skin cancer, would fix the tissue *in situ*. The fixative took as long as 24 hours to complete its task, but surgical removal was then accomplished without cutting through viable cancer, thereby eliminating the risk (fallacious though it was, in retrospect) of spreading the cancer. Tumor resection was followed by precise mapping and horizontal-section, microscopic examination of the removed tissue. The method of mapping of the resected tissue permitted very precise localization of any foci of residual tumor. Reapplication of chemical paste and further tissue removal followed until all tumor was removed. The technique proved remarkably efficient at removing even multiple recurrent tumors with cure rates that approached 98%.² Because of the chemical fixative used, the procedure became known as Mohs chemosurgery.

Mohs chemosurgery had few early proponents, primarily because of the difficulty in using the tissue fixative. The agent was painful when placed on the skin, not simply upon application, but for the entire time that the chemical was in contact with the skin. The pain was, at times, so intense that

patients required hospitalization for the sole purpose of pain relief. In addition, once the tumor was eradicated, there was a layer of fixed (i.e., dead) tissue that had to slough before reconstruction could be performed. The sloughing process took several weeks; therefore, virtually all Mohs surgical defects were left to heal by second intention, the only exceptions being those defects where functional considerations superseded, such as eyelid margin defects.

Over the course of the next 40 years, the Mohs procedure evolved. Clearly the most important modification was introduced by Theodore Tromovitch⁵ in the early 1970s. Tromovitch realized the illogic in using the fixative paste. He, therefore, eliminated its use during the procedure, while retaining the crucial aspects of precise mapping and horizontal sectioning of tissue. This modification meant that, since fresh tissue was being cut and processed by frozen section, multiple layers of tissue could be processed in one day, rather than one layer per day. The process of cancer removal was, therefore, significantly shortened. In addition, since no fixative was used, the final tumor-free defect could be immediately reconstructed. Despite Tromovitch's modifications, the same high cure rates of the original Mohs procedure were retained.

Within several years, Mohs surgeons had completely abandoned the use of the fixative, fully embracing the fresh tissue technique of Tromovitch. Accompanying the technical changes provided by the Tromovitch modification, there was an increased interest in the procedure. The number of fellowship-trained Mohs surgeons grew from fewer than 50 in 1970 to nearly 400 in 1997.⁶ Because of the confusion caused by the term *chemo* in the name chemosurgery, the name has been simplified to Mohs micrographic surgery. The term micrographic refers to the microscopic control and the making of the detailed maps.

Standard tumors and treatments

Most primary BCCs are successfully treated with standard modalities. Surgical excision⁷⁻⁹ and electrodesiccation and curettage (ED & C)¹⁰⁻¹³ have well-documented cure rates of 90% to 95%. Appropriate cryosurgical treatment,¹⁴⁻¹⁶ defined as using cryoprobes or appropriate freeze-thaw cycles, can result in similar cure rates.

These standard modalities, as diverse as they seem, all require the physician to visually estimate how much tissue to treat. This is done by first determining the clinical (i.e., visible) margins of a tumor and then estimating a safety margin of additional tissue to treat. All tumors have microscopic finger-like growths that extend variably beyond the visible margins (**Figure 1**). The safety margin, then, is

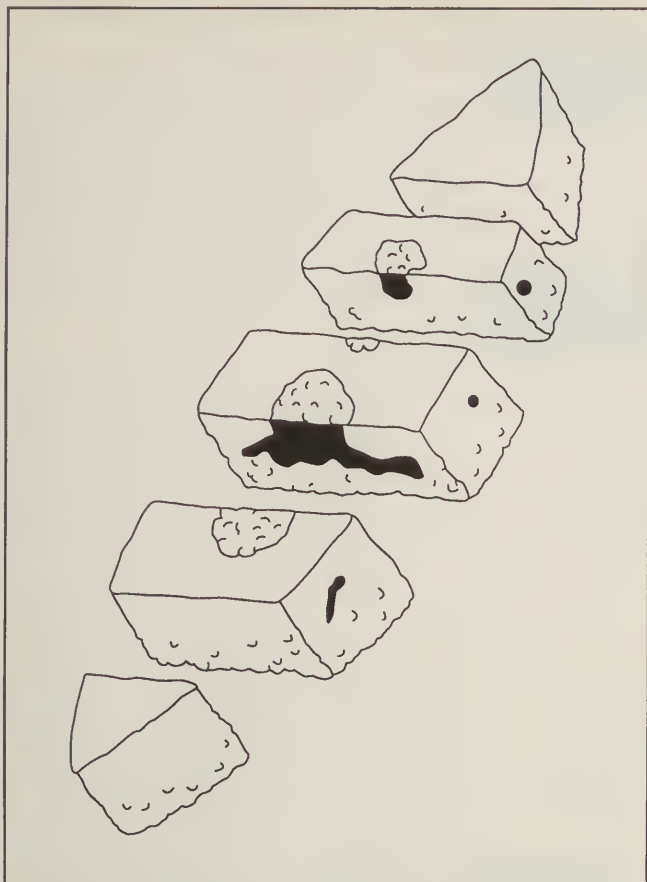


Figure 2. This hypothetical example demonstrating the vertical sectioning technique shows seemingly clear pathological margins but with three missed tumor foci at the margins.

intended to encompass within its boundaries those subclinical microscopic tumor extensions. If too small a safety margin is chosen, residual tumor is left behind and cancer recurrence is likely. If too great a safety margin is chosen, the potential for cure increases but at the expense of loss of normal, healthy tissue.

To complicate the problem of defining adequate safety margins is the realization that these cancers demonstrate eccentric (**Figure 1**), rather than symmetrical, growth characteristics.¹⁷ The microscopic fingers that grow from the main mass of tumor may extend many millimeters in one direction, while extending only one or two millimeters in another. When arbitrary margins of safety are chosen, there will be an obligatory loss of normal tissue from one or more sides of the visible tumor. For most tumors, for example, on the arm, back, or cheek, the removal of an additional few millimeters of tissue will likely have no adverse cosmetic or functional impact. However, for those tumors where tissue sparing is desired, for example, on the eyelid margin, nasal tip, and auricular helix, two millimeters of additional resection may make the difference between a simple closure and a more complicated skin graft or local flap. The precise

nature of the microscopic control offered by Mohs surgery eliminates the need to choose arbitrary safety margins. The procedure permits the preferential tracing out and removal of eccentric tumor extensions, thereby allowing for maximal tissue preservation.

Mohs micrographic surgery—technique

The principles upon which Mohs surgery is based, and which account for its well documented cure rates for the treatment of skin cancer, relate to the use of horizontal frozen sections to examine the tissue pathologically, the way tissue is excised and mapped, and the office-based nature of the procedure.

Horizontal sectioning of tissue. Before describing the technique of horizontal sectioning, it is important to contrast it with the universal standard of processing tissue that exists in pathology laboratories, that is, vertical sectioning of tissue.¹⁸⁻¹⁹ The concept of vertical sectioning can be demonstrated with the example of the standard elliptical specimen (**Figure 2**). When such a specimen is submitted for standard pathologic examination, slides are made from slices taken at each end of the specimen. In addition, and depending upon the size of the tumor, one or several additional sections are also taken from that portion of the specimen that contains the thickest tumor. While this sampling technique examines only about 0.1% of the entire surgical margin, it generally provides a reasonably good estimate of the actual margins. Pathologists understand the limitations of this sampling approach, which explains why most pathology reports contain statements such as, "margins are clear... in the sections examined," or "margins appear free of tumor."

With vertical sectioning, if there are thin microscopic fingers growing out from the main tumor mass that are not contained within the sampled sections, these tumor extensions will be missed and will serve as the nidus for tumor recurrence (**Figure 2**). This explains why a tumor will sometimes recur despite a negative pathology report. Unfortunately, there is a general lack of understanding among non-pathologists regarding this standard methodology for examining skin pathology. This lack of understanding causes great confusion for patient and surgeon alike when a recurrence is noted despite clear margins.

Mohs surgical specimens, by contrast, are processed using horizontal sections.²⁻⁵ Instead of the vertical incisions into the skin that are made by most surgeons when removing a specimen, Mohs surgical specimens are beveled at the edges (**Figure 3**). Taking advantage of the fact that skin is pliable, the beveled peripheral edges of the removed tissue can then be pressed down and flattened. This flattening

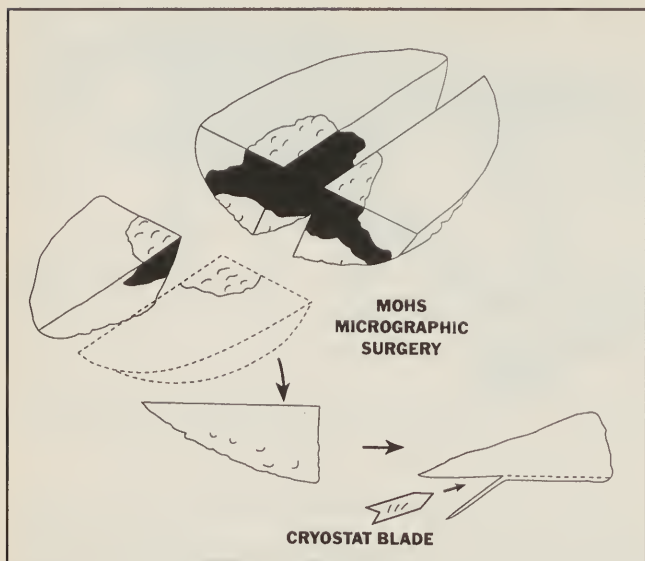


Figure 3. Beveled surgical specimen, the specimen then subdivided, flattened and horizontal section removed.

places the peripheral epidermal margins in the same plane as the deep margins; the entire interface between the specimen and the patient lies in a single plane. With careful frozen section technique by a well-trained Mohs laboratory technician, a single horizontal frozen section of tissue will permit evaluation of, not the estimated 0.1% of the surgical margins as is the case with vertical sections, but rather an examination that approaches 100% of the surgical margins. Small sub-clinical tumor extensions that would be missed by vertical sectioning techniques are less likely to be overlooked with this approach.

Precise tissue mapping. During surgery, the specimen to be removed and adjacent defect edges are carefully marked or notched *in situ* at the appropriate grid lines for the proposed map. The tissue is then removed, subdivided (in preparation for frozen sectioning) and color-coded (**Figure 4B**). A precise anatomic map is then drawn to include these subdivisions and dyed margins (**Figure 4D**). By creating a precise grid patterned map of the removed tissue, an exact evaluation of margins can be made.

This technique allows for the localization of residual tumor, often to within one or two millimeters. If there is residual tumor, these sites are marked on the map (**Figure 4D**), additional tumor containing tissue is excised (**Figure 4E**), the map is redrawn, and the additional tissue is processed and examined microscopically (**Figure 4F**). The process of tumor resection and pathologic examination continues until all tumor extensions have been excised and a tumor free margin is obtained (**Figure 4G**).

Office based Mohs micrographic surgery versus operating room margin controlled excision

When Mohs surgery is not available, patients with difficult tumors are often taken to the operating room for frozen section margin control with assistance from the pathology department. Aside from the problems associated with the use of vertical sectioning as described above, there are additional limitations associated with treating skin cancers in the operating room.

The primary problem involves time. The following scenario is standard for treatment of a skin cancer in the operating room. After the usual preparations are made, the first tumor resection is performed and the tissue is sent to pathology. The surgeon and staff then must stand around while the patient is waiting on the operating table, for a frozen section report from pathology. Because of the pressures of operating room time, the pathologist is often rushed to complete the sectioning and microscopic task. In teaching hospitals, such frozen section histology is often relegated to the least experienced resident rather than the most experienced histology technician. The report that is generated is usually fairly general and includes comments about the deep versus the peripheral margins, but generally does not describe specific sites of residual tumor. If tumor is noted to still be present at the surgical margins, the following scenario usually follows. Again because of time constraints, either due to the high cost of operating room time or because a case follows, a second and final specimen is taken. This specimen is also submitted, but this time for permanent sections. Since permanent sections take several days to be processed and read, the surgeon must close the defect without benefit of a final report from pathology. When the final report is available, if margins are still involved with tumor, the surgeon must deal with the dilemma of whether to take the patient back to surgery or to simply watch the site for a potential recurrence. This dilemma is particularly acute if the reconstruction was complex, as is often the case when dealing with tumors involving the eyelid, nose, and ear. In order to avoid this potential problem, the surgeon often feels obligated to take a larger margin of safety at the time of the second surgical resection.

Mohs surgery—an office-based procedure

In contrast, the following describes the typical skin cancer patient treated by office-based Mohs surgery. After the initial tumor resection, the surgical site is bandaged and the patient is allowed to wait with family in the surgical waiting room. This is far more comfortable for the patient, and also

frees the procedure room and staff. There are additional advantages to this approach. Since Mohs surgery is performed in the office, the external time constraints obligatory in the operating room do not exist. While most tumors are cleared with two to three stages, some cancers certainly require multiple stages to remove, as in the case of deeply invasive or wide-spread cutaneous tumor or when very conservative surgical margins are being utilized in order to preserve healthy tissue. The office-based nature of Mohs surgery allows for the time necessary for multiple surgical steps.

In addition, the Mohs laboratory is located in close proximity to the procedure rooms. This permits close communication between the surgeon and the histotechnician. Since the Mohs histotechnician performs only one task in the Mohs laboratory—that of cutting horizontal frozen sections—an appropriately trained and experienced technician can become quite proficient at providing complete sections (obligatory for adequate and complete examination of the complete surgical margins).

Indications for Mohs surgery

Mohs surgery is highly effective in the treatment of BCC and SCC. The initial papers by Tromovitch and Stegman in 1974 and 1978^{5,23} documented an overall cure rate of 97.2% in the treatment of 532 tumors. Mohs demonstrated a cure of 99.8% for 3466 cases of BCC followed for more than 1 year, and 98.8% of 822 cases of SCC followed for at least 1 year.²⁴ Similar cure rates have been published by other authors.²⁵⁻²⁶ While Mohs surgery is highly effective for the treatment of all BCC and SCC, the prime indication for the procedure is for the treatment of tumors at high risk for recurrence.²⁴⁻²⁷

The factors that increase the risk for recurrence include: tumor location,²⁸⁻³⁸ previous recurrence,³⁸⁻³⁹ size greater than 2 cm, and histologic aggressiveness.⁴⁰ Tumors with ill-defined clinical margins and those that arise in scars are also at high risk for recurrence.

Location. Tumors that arise on certain anatomic sites are well known to be at high risk for recurrence. These sites include the central triangle of the face which encompasses the perioral, perinasal, and periorbital areas, as well as the periauricular area (**Figure 5**). Eyelid tumors often represent a significant management problem. Conservation of tissue is often of critical importance in this location. Mohs surgery has been reported to offer a 99% cure for eyelid and canthal BCC. Periauricular tumors are particularly suited to the tissue conservation offered by Mohs surgery. Because of the precision of the margin control, cartilage can often be spared, preventing the need for full thickness reconstructions. The postauricular sulcus is particularly problematic, even after Mohs surgery, where a 16% recurrence rate has been noted. The nose represents the most common site for development of BCC and is associated with the highest risk for recurrence. The relative risk for recurrence after standard surgical procedures is 2.38.²⁶ Mohs surgery offers a 97% to 99% cure rate for such tumors.²⁴⁻²⁵ Since there is very little tissue on the nose available for reconstruction, tissue conservation is indicated.

Recurrent tumors. Recurrent tumors represent a prime indication for Mohs surgery. The fact that the tumor recurred suggests there were factors about the tumor that made it less responsive to standard treatment methods. Treatment of these tumors is also

complicated by scar tissue and anatomic alterations that often result from the prior treatment. Standard treatments

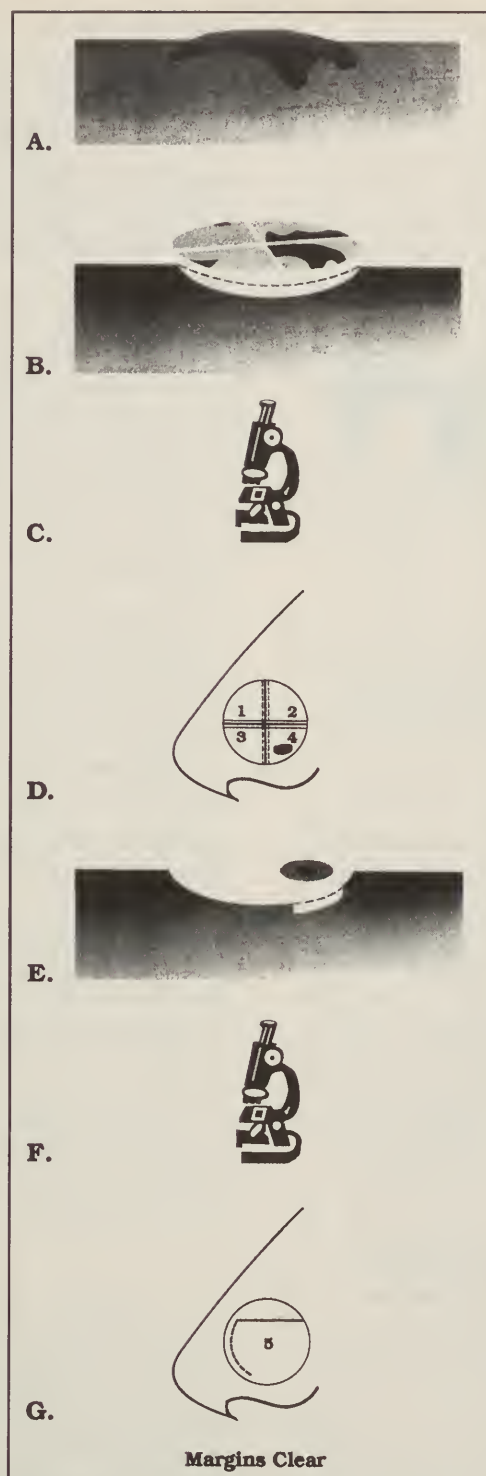


Figure 4. A. hypothetical tumor in cross-section; B. First layer of Mohs surgery with specimens subdivided into four sections; C. Microscopic examination of the tissue; D. The anatomic map (the nose in this example) with focus of residual tumor exactly marked; E. The surgeon returns to the patient and, using the map as the guide, removes only that focus of remaining tumor; F. the tissue is processed and examined; G. anatomic map demonstrating tumor free margin.

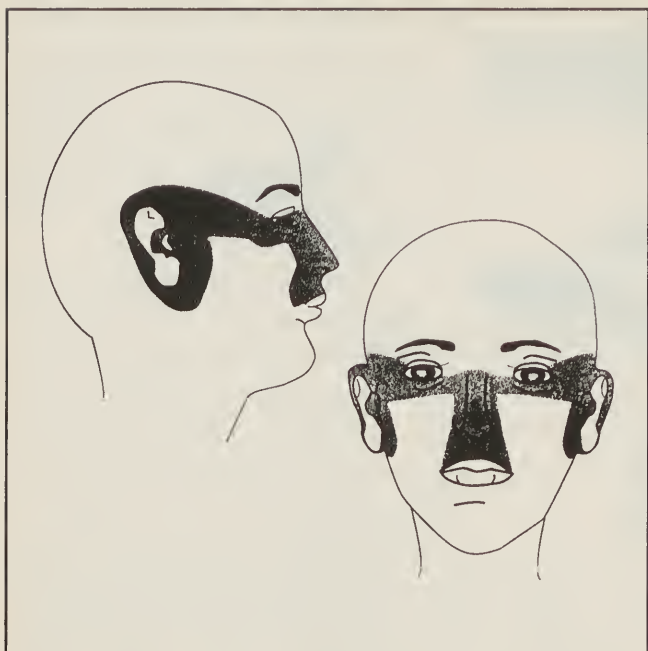


Figure 5. Anatomic sites where tumors are at high risk for recurrence including the central face, periauricular area, and temples.

have a dismal potential for cure with recurrence rates of between 20% and 50%.³⁸⁻³⁹ Despite these factors, Mohs surgery is only slightly less effective in the treatment of recurrent BCC than for primary tumors. Cure rates of 94% to 97% have been documented with Mohs surgery for recurrent BCC²⁴⁻²⁶ and slightly less for recurrent SCC.²⁷

Aggressive tumor subtypes. BCC of specific histologic subtypes have been shown to be more aggressive and are more likely to recur after standard therapy. These aggressive

subtypes include morpheaform (sclerosing), infiltrating, adenoid, and metatypical (basosquamous) types. Many pathologists do not appreciate the clinical importance of proper tumor subtype differentiation. Therefore, many pathology reports are simply signed out as BCC. It is, therefore, critical for any physician who treats BCC to be certain that their pathology reports accurately report tumor subtypes.

Incompletely excised tumor. Incompletely excised tumors deserve specific mention. The overall recurrence rate for incompletely excised tumors is 35%. Why 100% of such tumors do not recur is the subject of much speculation, but this somewhat lower rate of recurrence has been cited as the reason why incompletely excised tumors can be watched rather than retreated. Unfortunately in 1997, a potential recurrence rate of 35% is not acceptable. The rate of recurrence is much higher,⁴¹ unfortunately (reportedly as high as 82%), for recurrent tumors in the perioral, periorbital, and perinasal areas. Mohs micrographic surgery should be strongly considered for any incompletely excised tumor.

Other indications. The precise margin control that exemplifies Mohs surgery is useful only for tumors that grow by contiguous extension. Tumors that exhibit skip areas are not amenable to the procedure. **Table 1** lists other tumors for which Mohs surgery has been shown to be effective.

Multispecialty approach to complex tumors. The treatment of complex and often multiple recurrent skin cancers requires that the Mohs surgeon work within a multispecialty framework that includes general plastic, oculoplastic, and facial plastic surgeons, as well as radiation, surgical, and medical oncologists.⁴² Such a relationship allows for the most effective management for patients with multiple recurrent or very large tumors.

Summary

In summary, Mohs micrographic surgery is a unique surgical and pathologic approach for the treatment of skin cancer, particularly for the treatment of tumors at high risk for local recurrence. The office-based surgical procedure combines the benefits of precise microscopic control with normal tissue preservation and has the additional benefit of being cost-effective.

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Table 1. Adapted from the Guidelines of Care for Mohs Micrographic Surgery, the American Academy of Dermatology, 1995.²¹

- ▶ Verrucous carcinoma
- ▶ Microcystic adnexal carcinoma
- ▶ Keratocanthoma (aggressive, recurrent, or mutilating)
- ▶ Dermatofibrosarcoma protuberans
- ▶ Atypical fibroxanthoma
- ▶ Malignant fibrous histiocytoma
- ▶ Sebaceous carcinoma
- ▶ Extramammary Paget's disease
- ▶ Apocrine carcinoma of the skin
- ▶ Leiomyosarcoma
- ▶ Adenocystic carcinoma of the skin
- ▶ Erythroplasia of Queyrat
- ▶ Merkel cell carcinoma

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Nonmelanoma skin cancer

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ABSTRACT: *Nonmelanoma skin cancer is the most common cancer in the United States, and its incidence is increasing dramatically. Although mostly caused by sun exposure, less common causes are also discussed. Several types of basal cell carcinomas are described, as well as squamous cell carcinoma. While these carcinomas rarely cause death, they can be locally destructive, and removal can cause significant disfigurement and functional impairment. The value of early detection is emphasized.*

Basal cell carcinomas and squamous cell carcinomas are the most common malignancies in Caucasians. It is estimated there are 800 000 new cases (1/3 of all new cancers) each year in the United States.^{1,2} These carcinomas rarely cause death, but they can invade locally and lead to significant destruction. Due to their location, usually on sun-exposed skin, removal can cause significant disfigurement. The actual incidence of these carcinomas may be three times the above stated figure, and this incidence is increasing dramatically.³ This increase is largely due to greater exposure to the sun. More leisure time and more outdoor recreational activities, such as golfing, biking, tennis, and hiking, greatly increase exposure. Easier access to sunny destinations during the winter and the use of tanning booths have increased year-round exposure. The reduced ozone layer may also be contributory.

Because these carcinomas are due to an accumulation of sun exposure, the risk is greatest among those aged 60 to 65⁴ who have had both years of exposure and a prior lack of knowledge regarding the harmful effects of the sun. However, these carcinomas are commonly seen in patients in their twenties and thirties. Evidence suggests the sun's role in causing these carcinomas. Most (2/3) occur on sun-exposed skin.⁴ Outdoor workers have significantly higher incidences of nonmelanoma skin cancers. The risk is greater at lower latitudes, so a person living in Arizona is more than twice as

Basal cell carcinomas

Most nonmelanoma skin cancers are basal cell carcinomas. Fortunately, basal cell carcinomas almost never cause death. The rare cases of metastases occur in very large tumors, neglected tumors, or recurrent tumors. As mentioned, two-thirds of basal cell carcinomas occur on sun-exposed skin.

Basal cell carcinomas can take several different clinical appearances. The most common type is nodular (Figure 1). Nodular basal cell carcinomas are translucent-pink papules, often with overlying telangiectasias and central depression. Like all basal cell carcinomas, they grow slowly and can be present for months or years before the patient seeks medical attention. As the nodular type grows, it can become ulcerative and very destructive (Figure 2). Nodular basal cell carcinomas can be pigmented (Figure 3), confusing the diagnosis,

but not altering clinical implications.

Another common type of basal cell carcinoma is superficial. These are discrete, red scaling plaques (Figure 4). This type can be mistaken for a patch of nummular eczema, psoriasis, or Bowen's disease (squamous cell carcinoma *in situ*).

A less common variant of basal cell carcinoma is the morpheaform or sclerosing type (Figure 5). Difficult to diagnose because it can be barely perceptible, this type tends to be a white to pink, slightly depressed, poorly defined lesion that can resemble a scar. The histologic borders of a morpheaform basal cell carcinoma extend well beyond the clinically apparent



Figure 1. Basal cell carcinoma: nodular type

likely to develop a cutaneous carcinoma as someone living in Minnesota.^{5,6} Similarly, skin pigmentation is an important factor. Persons who are fair-complected and who burn easily are at greater risk than those with dark complexions. Nonmelanoma skin cancer is uncommon in African-Americans and Hispanics in whom increased amounts of melanin provide protection.

Other factors are also involved in the development of nonmelanoma skin cancers. Patients with certain genetic disorders such as xeroderma pigmentosum, in which the skin fails to repair sun-induced DNA damage, have a very high incidence of skin cancers, beginning at a young age. Immunosuppressed patients, such as those with renal transplants, have an increased risk. Therapeutic radiation and arsenical exposure are also associated with increased risk. Scars from vaccinations or burns or chronic ulcers (i.e., venous stasis ulcers) are sites with known increased risk of carcinoma. Certain strains of human Papillomavirus can also lead to squamous cell carcinomas.



Figure 2. Basal cell carcinoma: ulcerated



Figure 3. Basal cell carcinoma: pigmented



Figure 4. Basal cell carcinoma: superficial type

lesion, and its removal requires wider margins of excision than other clinical variants.

Diagnosis and treatment of a basal cell carcinoma includes a biopsy followed by appropriate surgical excision. Surgical excision is accomplished by either curettage and electrodesiccation or excision with suture repair. Mohs micrographic surgery is an office-based technique, involving tissue mapping and frozen sections, and is used for complicated and/or recurrent tumors.

Radiation therapy is also used for some skin cancers. However, while ionizing radiation renders good cure rates and avoids surgery, it also requires daily treatment sessions lasting typically four to six weeks. Additionally, the early cosmetic results are excellent but tend to form noticeable scars as time progresses. This technique should be reserved for inoperable tumors or for those patients who either will not tolerate or refuse surgery.

Follow-up of patients diagnosed with basal cell carcinoma is critical. These patients are at risk for recurrence and for devel-

opment of new primary carcinomas in other locations. Most recurrences occur within two years but may not appear for ten years. Recurrence depends on size, location, type, and the method of treatment used.^{7,8,9} Recurrence is most common for the morpheaform type.^{10,11} A five-year prospective study showed that of patients treated for basal cell carcinoma, 33% developed a second carcinoma in another location by the end of the second post-operative year, and only 27% of these were detected by the patient.^{12,13} Another study showed 50% of patients with a history of nonmelanoma skin cancer will develop a new skin cancer within five years.^{14,15} It is, therefore, recommended that these patients be examined every six months for two years and yearly thereafter.

Squamous cell carcinomas

The second most common nonmelanoma skin cancer is squamous cell carcinoma. Although basal cell carcinomas are much more common than squamous cell carcinomas, the ratio of squamous cell carcinomas to basal cell carcinomas increases with greater sun exposure. Thus, the incidence of squamous cell carcinoma, relative to basal cell carcinoma, is increased in Texas and decreased in Boston. Most deaths from nonmelanoma skin cancer are due to squamous cell carcinoma. The risk of metastasis is dependent upon the site of the cancer and whether there is a predisposing injury. Squamous cell carcinomas that develop on sun-exposed areas have a metastatic rate of less than 1%,¹⁶ whereas those arising on mucosa (lower lip) have a



Figure 5. Basal cell carcinoma: morpheaform type



Figure 6. Actinic keratoses



Figure 7. Actinic keratosis

metastatic rate of 16%.¹⁷ For squamous cell carcinomas arising in radiation sites, scars, and ulcers, the metastatic rate is between 18% and 30%.^{18,19,20}

Like basal cell carcinomas, most squamous cell carcinomas occur on sun-exposed areas. About 80% occur on the head, neck, and arms. Those occurring on non-exposed skin are often due to other causes, including radiation, immunosuppression, scars, or chronic ulcers. Squamous cell carcinoma can develop either *de novo* or, uncommonly, from an actinic keratosis. Actinic keratoses are precancerous, occasionally progressing to squamous cell carcinomas. They are small, red, scaling, often keratotic lesions. They are frequently multiple in a given patient



Figure 8. Squamous cell carcinoma

(Figures 6 and 7). The treatment of actinic keratoses with superficial destructive means such as cryotherapy can prevent their progression into a carcinoma.

Squamous cell carcinomas typically present as red, scaling, crusted, or ulcerated nodules. They often can be indistinguishable from basal cell carcinomas (Figures 8, 9, and 10). Squamous cell carcinomas have more potential than basal cell carcinomas to be locally destructive and to metastasize via lymphatics or blood vessels, particularly those on the ear, temple, lips, and genitalia.

Bowen's disease is a superficial form of squamous cell carcinoma that presents as a solitary red, scaling plaque that can be difficult to distinguish from psoriasis, nummular eczema, or the superficial type of basal cell carcinoma (Figure 11). Bowen's disease can progress, if untreated, to invasive squamous cell carcinoma.

Keratoacanthomas are nodular tumors with a central crust or crater (Figure 12). They can appear clinically identical to squamous cell carcinomas. Keratoacanthomas, unlike nonmelanoma skin cancers, have a relatively acute onset and may resolve spontaneously over many months. Because they can be indistinguishable from squamous cell carcinomas, both clinically and histologically, they are best treated as low grade squamous cell carcinomas and surgically excised.

Diagnosis of a squamous cell carcinoma is made by biopsy. Treatment is similar to that for basal cell carcinoma with surgical excision. As stated earlier, there is a risk for both recurrence and development of new skin cancers in patients diagnosed with an initial squamous cell carcinoma. This is especially true during the first five years postoperatively.

Prevention

Although rarely life-threatening, nonmelanoma skin cancers can be locally destructive. The scars produced by their removal (even of nondestructive tumors) can be disfiguring. Furthermore, carcinomas affecting important struc-

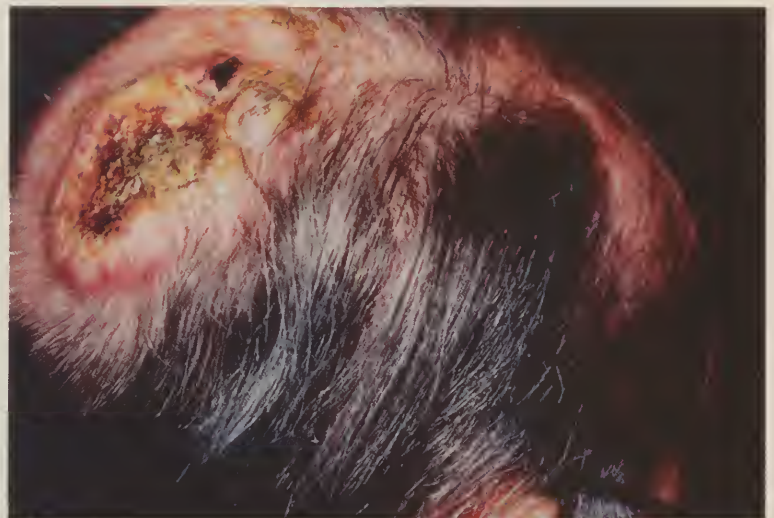


Figure 9. Squamous cell carcinoma



Figure 10. Squamous cell carcinoma



Figure 11. Bowen's disease (squamous cell carcinoma *in situ*)



Figure 12. Keratoacanthoma

tures such as eyelids can cause functional impairment. Early detection, when tumors are small and uncomplicated, can reduce disfigurement and functional impairment. By reducing the amount of sun-exposure, one can greatly reduce the risk of nonmelanoma skin cancers. Early education is critical.

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Atypical mole syndrome: A brief overview for the primary care physician

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ABSTRACT: *Early detection of melanoma and identification of potential markers or precursor lesions can substantially improve survival. Several risk factors have been identified in the pathogenesis of this potentially lethal cancer. Numerous reports in the literature have confirmed a subset of persons with an increased risk of developing melanoma. These patients are identified by a distinctive clinical phenotype depicted by unusually appearing melanocytic nevi (moles) in association with an increased number of total body nevi. They may have a family history of atypical moles or melanoma. In order to facilitate the recognition of such individuals by the non-dermatologist, a brief overview and salient features of the atypical mole syndrome are presented.*

Introduction

While initial reports of atypical (dysplastic) nevi occurring in the setting of familial melanoma may have been described earlier than the turn of the century, it was not until 1978 that Clark and associates described a distinctive syndrome scientifically linking the two together.¹ In their landmark paper, they studied six family pedigrees affected by familial melanoma and distinctive melanocytic nevi. These nevi, possessing characteristic clinical and histological features, were called "B-K moles," named after two young patients in their study. The syndrome was characterized by the presence of large irregular nevi with variegated color and a palpable dermal component. The number of B-K moles in an affected individual varied, from less than 10 to greater than 100, with striking heterogeneity from one lesion to another. The nevi were a phenotypic marker for members of the pedigree at risk for developing melanoma. They were also shown to be precursor lesions for melanoma.



Figure 1. Back of a 40-year-old man showing prominent mole pattern with atypical nevi.



Figure 2. Back of a 29-year-old woman with atypical nevi and a prior invasive melanoma.

Subsequently, atypical nevi were proven markers for nonfamilial melanoma.²

The term “dysplastic” nevus has now replaced “B-K mole” in the literature.^{2,3} More recently “atypical” nevus has been proposed.⁴ Regardless of the terminology, most dermatologists recognize this distinct clinical entity. The goal of this paper is to familiarize primary care physicians with the main features of atypical nevi, so they can identify patients at risk for developing melanoma. Placement of such individuals in surveillance programs allows for the detection of melanoma at an earlier stage, where the prognosis is excellent after surgical removal with adequate margins.

Clinical features

The clinical features of atypical nevi have been reviewed in the literature by several authors.^{3,5} The syndrome may occur as a sporadic or familial trait.² The familial form is thought to be inherited in an autosomal dominant fashion with variable penetrance. The clinical spectrum varies and is somewhat controversial, but most affected individuals have numerous (usually >50) melanocytic nevi, some of which display an atypical appearance (**Figures 1,2**). Atypical nevi are usually large, ranging in diameter from 6 mm to 15 mm (**Figure 3**); however, smaller nevi may also show the characteristic morphology. These nevi often have irregular borders and margins that fade imperceptibly into the adjacent skin (**Figure 4**). Their color is commonly variegated with a haphazard mixture of tan, brown, and pink. They are flat or slightly elevated with a mamillated surface (4). In

summary, they share some of the same features of malignant melanoma, and often a biopsy is indicated for a definitive diagnosis. Hypertrichosis and ulceration are never present. Pruritus (itching) and bleeding are not features of atypical nevi and are more suggestive of malignancy.

In addition to having numerous nevi, the distribution of atypical nevi favors the trunk and upper extremities. Affected persons will often have nevi in sun-protected areas, such as the scalp, buttocks, female breasts, and palms and soles.⁶ In fact, the occurrence of nevi in these unusual locations in prepubertal children may often be the first clinical indication of the trait.⁷ Similar to common acquired nevi, atypical nevi are not present at birth. They gradually appear during early childhood analogous to their common nevi



Figure 3. Abdomen with a cluster of atypical nevi showing heterogeneity of nevi.

counterparts. By contrast, however, at the time of puberty, the characteristic features of atypical moles begin to appear. Persons with atypical moles may continue to develop new nevi throughout their adult life, in contrast to unaffected persons, where nevi cease to occur after the age of 30.⁸

Histological features

The histologic criteria for the diagnosis of atypical nevi remains controversial, despite a recent National Institutes of Health (NIH) Consensus Conference on the diagnosis and treatment of early melanoma.⁹ Furthermore, dermatopathologists have yet to agree upon universally acceptable nomenclature for the atypical mole, since the term "dysplastic nevus" has been entrenched throughout the literature for the past two decades. The published criteria from the consensus conference are listed as follows: architectural disorder with asymmetry, subepidermal fibroplasia, and lentiginous melanocytic hyperplasia with spindle or epithelioid melanocytes aggregating in nests of variable size and forming bridges between adjacent rete ridges. Melanocytic atypia may also be present in varying gradation. The dermis may be infiltrated by lymphocytes and

the "shoulder" phenomenon (intraepidermal melanocytes extending beyond the main dermal component either singly or in nests) may be present.⁹ In view of the lack of agreement on histologic criteria and the current semantic debate, many dermatologists and investigators do not require biopsy confirmation of atypical moles. Although, practically speaking, most dermatopathologists would likely agree upon the histologic criteria of a nevus with moderate-to-severe cellular atypia.

Clinical management

Patients with atypical moles should have routine skin examinations beginning around puberty or enroll in a surveillance program at a major referral center's pigmented lesion clinic. The recommended frequency of examinations depends on personal or family history of melanoma and the number of atypical moles, but generally range from every 3 months to 12 months.^{10,11} Examination should not be limited to exposed areas and should include careful inspection of the scalp, genitalia, and acral regions.¹² Patients may also be at increased risk for developing intraocular melanomas; therefore, a baseline ophthalmologic examination should be obtained with appropriate follow-up as warranted.¹³ Family members should also be examined, even if a history of atypical moles is not elicited.⁷ Clini-



Figure 4. Close-up of a biopsy-proven atypical nevus displaying indistinct border.

cally, photography is an extremely useful tool for the identification of changes in nevi. At our institution, baseline cutaneous photographs with close-up views of the most clinically atypical moles are taken during the initial examination, and a set of photographs are provided to the patient to aid in their own assessment. Any lesion with documented change (i.e., the appearance of a dark brown or black spot, a color change, increase in size) should be removed in its entirety and sent for histopathologic examination.⁹ Nevi in which the patient has noted a change should also be removed.

Patient education and awareness can not be overemphasized. Patients should start by becoming familiar with their own skin and by performing monthly self-examinations. Patient guidelines have been published on methods of self-examination and early signs of melanoma and are available from a number of organizations, including the American Academy of Dermatology, Skin Cancer Foundation, and The American Cancer Society.

Sunlight has been implicated in the pathogenesis of malignant melanoma and the induction of melanocytic nevi in patients with atypical moles.^{14,15} Patients should use sunscreens with a high Sun Protection Factor (SPF) of 15 or greater daily, even on overcast or cloudy days. Re-application of sunscreens every 2 hours to 3 hours, particularly after swimming or exercising, should be strongly encouraged. Sun avoidance and sun-protective clothing are also significant methods of minimizing ultraviolet exposure.

Summary

Patients with atypical moles are a distinct group with an easily recognized clinical phenotype. Their risk of developing malignant melanoma is markedly increased over the general population, particularly if there is a family history of melanoma. Clinical management of such patients should focus on patient education, self-examination, and routine complete skin examinations.

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Diagnosis of pigmented skin lesions aided by epiluminescence microscopy

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ABSTRACT: *Early diagnosis of superficial melanoma (Clark Level I, II) remains the best approach to reduce the death rate from this malignant neoplasm. Today's well-informed patients understand the need to have changing moles evaluated. However, many benign pigmented lesions that undergo changes in appearance do not require excision. Epiluminescence microscopy can facilitate the differential diagnosis of cutaneous pigmented lesions and help determine which of these require biopsy.*

Introduction

Given the dramatic increase in the incidence of malignant melanoma,^{1,2} there is an urgent need to prevent an equally dramatic rise in the melanoma death rate. Until more effective therapy becomes available, the best approach toward reaching this goal is to enhance our diagnostic ability for early (Clark Level I, II) melanoma. (Clark Level I tumors have malignant cells confined to the epidermis; Level II penetrate into the papillary dermis only.) Certain diagnosis of a small, 1 mm to 3 mm, melanoma can be difficult relying solely upon visual inspection. The inability to differentiate between benign, suspicious, and malignant pigmented skin lesions may lead to excessive or unnecessary biopsies. Since one of the most well-known warnings of cancer is a change in a mole, there may be a tendency for patient and physician alike to desire removal of any pigmented lesion that has undergone change in size, shape, or color. However, dermatologists know that many benign pigmented lesions, such as seborrheic kera-

toses, lentigines, and common benign nevi, change size, color, and shape over the years and do not require excision.

Description of the technique

Epiluminescence microscopy (ELM), also referred to as dermatoscopy or skin surface microscopy, is a valuable diagnostic tool for the examination of pigmented skin lesions.³ The origins of this technique date back more than a century to when magnification was used to examine the nail fold capillaries of lupus erythematosus, and lupus vulgaris was examined for apple jelly coloration by pressing a glass slide on the lesion. In skilled hands, the regular use of ELM has been documented to increase the diagnostic accuracy for early melanoma and decrease the total number of biopsies taken. ELM does not eliminate all ambiguity in differentiating between the benign nevus and the malignant melanoma, but it improves the diagnosis of clinically borderline lesions by approximately 20% to 30%.⁴

ELM involves the use of a 10X magnifying illuminated dermatoscope, an instrument resembling an otoscope. A flat glass surface is placed firmly against the skin, which has been covered with immersion or mineral oil. The use of oil improves visualization by permitting light to penetrate deeper into the skin, rather than being reflected off it. Upon first use of ELM, one is impressed that the familiar visual criteria for the differential diagnosis of pigmented lesions are gone and that new ones must be learned. Once new diagnostic criteria are employed, melanoma can more readily be differentiated from common benign nevi, seborrheic keratoses, lentigines, angiomas, pigmented basal cell carcinomas, and other lesions. The differential diagnosis between atypical nevus and early melanoma is not absolute with ELM, but the presentation of this dilemma represents a clear indication for biopsy.

Diagnostic criteria – ABCD

The diagnostic criteria employed by ELM are also outlined in ABCD format.⁵ A represents asymmetry in color or shape of the lesion. B indicates evaluation of the border of



Figure 1A. This medium brown papule has a poorly defined light brown periphery.

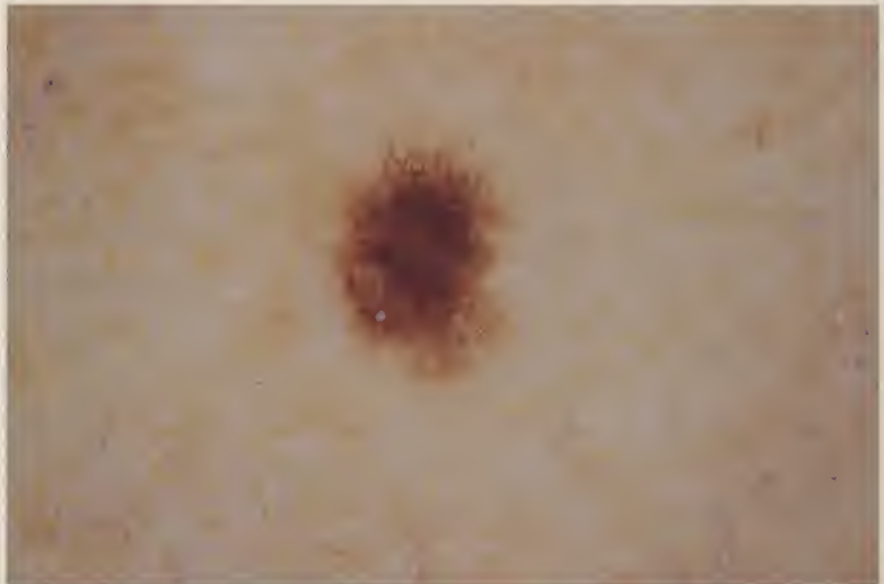


Figure 1B. With epiluminescence microscopy (ELM) both the light and medium brown pigment networks are seen to be intact. The pigment network fades into the surrounding skin with no abrupt cutoff. Small dots of pigment are located centrally within this benign compound nevus.

the lesion for abrupt cutoff of the pigment pattern, as opposed to the gradual thinning of the lesion into the periphery seen in benign lesions. C is for the evaluation of color; malignant melanoma may have up to six colors (i.e., white, red, light brown, dark brown, slate blue, and black). D indicates the presence of differential structures within the melanoma. These include alteration of the normal pigment network, the presence of pigmented dots, globules, and radial streaks, and bluish-white structureless areas (often referred to as “milky veils”).

The observed color depends on the histological location of the pigment within the skin. If melanin is in the upper



Figure 2A. This 2 mm brown macule has been present for 2 months on the anterior surface of the right thigh. An eccentric dark speck of pigment is present.



Figure 2B. With ELM, the eccentric brown-black globule is accentuated. Radial streaming is present in this melanoma *in situ*.

epidermis, it will be seen as black. If it is at the junction of the dermis and epidermis, it will be brown. It will be blue if it is located deep in the reticular dermis. In benign nevi, brown pigment is typically seen as a mesh or net with delicate strands and small spaces between the strands. A network with thicker and darker strands and wider spaces between them may indicate a dysplastic or atypical nevus or a melanoma.

Brown globules indicate nests of nevus cells in the lower epidermis or upper dermis. In benign lesions, they tend to be regular in size and shape and distribution, while in atypical nevi and melanomas, they are irregular in size and scattered

within the lesion. When black dots are observed, indicating melanin within the upper epidermis or stratum corneum, they tend to be clustered in the center in benign lesions, but appear more frequently and at the periphery in malignant ones. Streaks of pigment from the center of the lesion to the periphery (radial streaming) indicate the pigment network is no longer intact and the lesion is likely a melanoma. Depigmentation occurs within regressing benign and malignant lesions, but is most often in the center in benign lesions and irregularly scattered toward the periphery in malignant ones.

When benign melanocytic nevi are examined by ELM, a homogenous, symmetrical, fine mesh network of pigmentation is seen. The network does not end abruptly but gradually blends into the surrounding skin (Figure 1). In particular, radial streaming and large numbers of eccentric black dots are not seen. Atypical or dysplastic nevi have darker pigment networks that end abruptly at the periphery of the lesion. Depigmentation may be present but not radial streaming or large numbers of peripheral black dots. The ELM appearance of melanomas varies considerably, reflecting the variety of presentations of melanoma seen clinically, that is, a small *in situ* superficial spreading melanoma will differ from a large, deep nodular melanoma (Figures 2 and 3). The worrisome ELM features of melanoma as mentioned above include radial streaming, which may occur in varying colors, peripheral black

dots, and disruption of the pigment network at the periphery of the lesion. If these features are present in a lesion, immediate excision is indicated.

Non-melanocytic lesions, such as seborrheic keratoses, hemangiomas, and pigmented basal cell carcinomas, can be differentiated from nevi and melanomas by ELM. Seborrheic keratoses commonly lack a pigment network; they have pseudo-follicular openings and, most characteristically, they have punctate whitish-yellow spots indicating intraepidermal horny pseudocysts. Hemangiomas also lack a pigment network and have sharply outlined, reddish-black saccules centrally and small capillaries peripherally. Pigmented basal cell carcinomas

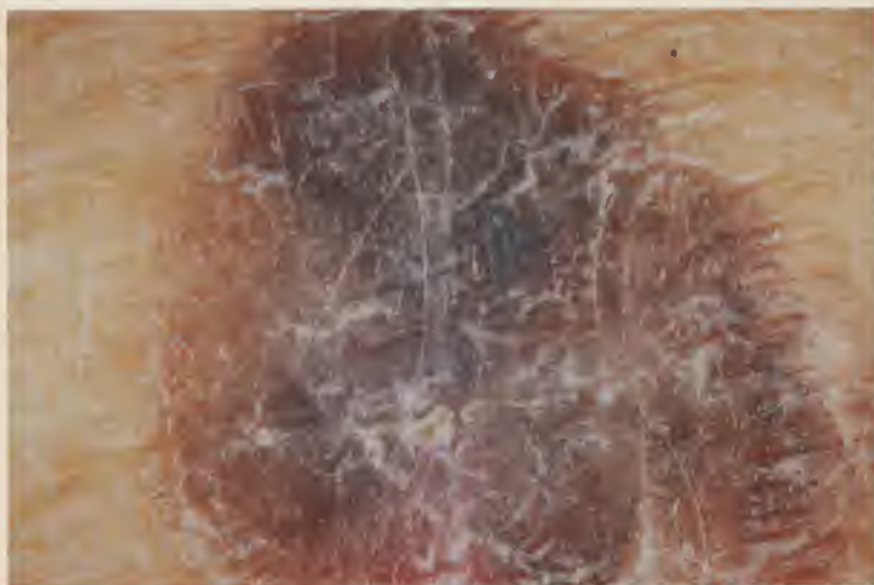


Figure 3A. This scaly, medium to dark brown lesion is not clinically diagnostic of melanoma.

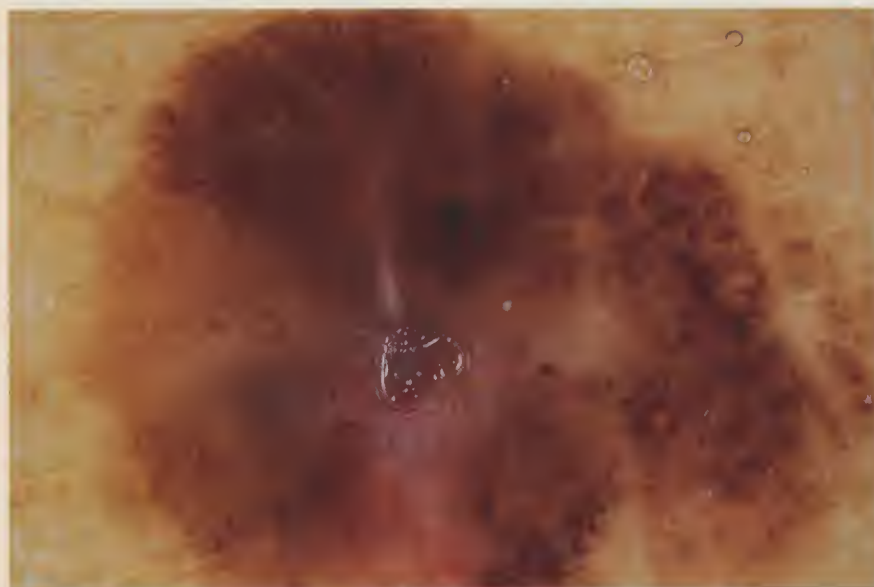


Figure 3B. With ELM, multiple colors can be appreciated. These include areas of dark and light brown, a black globule of pigment (above and to the right of the central air bubble), and a central bluish-white amorphous area (milky veil) underneath the bubble. The pigment network in the dark brown areas is coarse with wide spaces and abruptly terminates at the periphery. These findings are diagnostic of melanoma.

have no pigment network, but they usually show a maple leaf pigment pattern and telangiectatic capillaries.

Conclusion

ELM represents a significant step forward in the early diagnosis of melanoma. Its use is comparable to the gynecologists use of colposcopy for cervical dysplasia and neoplasia. ELM is limited when lesions lack pigment, such as in the non-pigmented benign intradermal nevus and in the amelanotic melanoma. Those interested in pursuing the use of the dermatoscope would benefit from reviewing dermatoscopic articles^{3,4} and atlases⁵ and from participating in seminars.

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FOR YOUR PATIENTS

The Cancer Research Institute and Cancer Care, Inc. recently launched "The Melanoma Initiative." In addition to enhancing public awareness and developing research programs, this initiative includes a nationwide system of support services to help patients cope with the emotional, social, and financial burdens of melanoma. Counseling and practical assistance; telephone support groups and educational seminars; a "buddy" program; guidance on medical services; a referral program for locating community resources; and guidance on home care, transportation, child care, and financial assistance are all available at no charge. These patient outreach services are available through Cancer Care's toll-free national counseling line, 1-800-813-HOPE, and its web site at <http://www.cancercareinc.org>.

The surgical management of invasive primary melanoma: An update

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Introduction

In the past four decades, the incidence of melanoma has increased worldwide and tripled in the United States.^{1,2,3} An estimated 32 000 new cases of melanoma and 6500 deaths related to melanoma are reported each year in the United States.⁴ It is estimated that by the year 2000 the lifetime risk of developing melanoma will be 1 in 90 for Caucasians living in the United States.^{5,6} Five-year overall survival is approximately 81%. This is greatly improved from a five-year overall survival of 49% in the 1950s. However, the total mortality attributable to melanoma has increased to 150% over the past four decades because of the increased incidence.⁷

For newly diagnosed primary melanoma, the initial treatment is surgery. The role of the surgeon in the treatment of primary melanoma is to remove the primary lesion in such a way that its chance of local recurrence is minimized. Surgeons also stage and manage the regional nodal basin. The goals of treatment are to prevent local recurrence and to improve survival whenever possible. This paper will discuss surgical management of invasive primary melanoma.

Management of the primary lesion

Every patient with a newly diagnosed invasive melanoma requires an excision of the primary lesion. Over the last two decades, the standard recommendations for excision margins have changed. In the past, resection margins of up to 5 cm were felt to be necessary for local control of primary melanoma. Prospective studies have now demonstrated that narrower margins are safe for most melanomas.

The World Health Organization (WHO) Melanoma Group randomized 612 patients with invasive melanoma less than 2 mm in thickness to wide local excision using 1 cm margins versus 3 cm margins. Only four

patients had a local recurrence at the first site of melanoma recurrence; all of them had a melanoma 1 mm to 2 mm in thickness and had received a 1 cm excision. This study demonstrated that a 1 cm margin of excision is safe for melanomas <1 mm in thickness.⁸ Subsequently, the Intergroup Trial for intermediate thickness melanoma reported results of a prospective study that randomized 486 patients with primary melanomas 1 mm to 4 mm in depth to excision margins of 2 cm versus 4 cm. The local recurrence rates and overall survival rates were equivalent in the two groups at a median follow-up of six years. The patients whose melanomas were excised using 2 cm margins were much less likely to require skin grafting for coverage and had shorter hospital stays.⁹

The current recommendations for wide local excision margins are summarized in the table below. For lesions less than 1 mm in depth, a 1 cm wide local excision is considered appropriate. For lesions 1 mm to 4 mm in depth, a wide local excision using 2 cm margins is appropriate. For lesions greater than 4 mm in depth, appropriate excision margins have not been subjected to prospective studies. Excision margins in the thick melanomas should certainly be no less than 2 cm. Typically 2 cm to 3 cm excision margins are felt to be minimally acceptable for thick primary melanomas.

Management of the regional nodal basin

The regional nodal basin is the most common site of metastatic disease in patients with primary melanoma. The risk of nodal metastases is related to the depth of the primary. At the time of presentation, patients with primary lesions less than 1 mm in depth have a risk of identifiable synchronous nodal metastases of 2% to 10%. For patients with intermediate thickness lesions (1 mm to 4 mm), the risk of identifiable synchronous nodal metastases is approximately 20% to 25%. For patients with thick primary melanomas (greater than 4 mm), that risk is 50% to 60%.

Nodal metastases are of tremendous prognostic importance for patients with newly diagnosed melanoma.¹⁰ Nodal metastases remain the most powerful factor for predicting survival. Survival is associated with the presence of nodal metastases and also with the number of positive lymph

nodes. Five-year survival for patients with pathologically negative lymph nodes is approximately 90%. For patients whose lymph nodes are clinically negative but pathologically positive, the five-year survival is approximately 50% to 60%. For patients with clinically and pathologically positive lymph nodes at the time of presentation, five-year survival is approximately 15% to 20%. The number of positive lymph nodes is associated with survival. For patients with one positive lymph node, the five-year survival is approximately 50%. For patients with 2 to 4 positive lymph nodes, five-year survival is 20% to 40%, and for patients with five or more positive lymph nodes, the five-year survival is less than 20%.¹¹

For years surgeons have debated the benefit of elective lymph node dissection (ELND) for patients with newly diagnosed melanoma. ELND refers to an anatomic lymphadenectomy of the primary draining nodal basin when that nodal basin is clinically negative. The theoretic advantage of this procedure is to resect nodal disease at the earliest possible time. The disadvantage is that approximately 80% of patients with intermediate thickness melanoma (1 mm to 4 mm) have negative lymph nodes at presentation, and therefore presumably accrue no benefit from this procedure. At the time of the diagnosis of a primary melanoma, a patient with a clinically negative nodal exam might have: 1) the primary only with negative nodes and no distant metastases; 2) a primary melanoma with nodal metastases but no distant metastases; or 3) the primary melanoma with or without regional nodal metastases, but with distant metastases. Theoretically, only the second group could potentially benefit from ELND.

The morbidity of ELND can be quite troublesome to the patient. Patients undergoing lymphadenectomy incur the expense and inconvenience of surgery and a one- to two-day hospital stay. Postsurgical complications of wound infection, dehiscence, or poor healing occur in 20% to 25% of patients.^{12,13} Symptomatic lymphedema occurs in 20% to 25% of patients undergoing inguinal lymphadenectomy, even with appropriate prophylactic measures.¹⁴

The question of ELND has been debated particularly with respect to intermediate thickness (1 mm to 4 mm) melanoma. Prospective randomized trials of elective lymph node dissection versus observation in this group have failed to show an overall survival advantage. Early studies involving patients with melanoma of any thickness reported by Veronesi¹⁵ and by Sims¹⁶ have been criticized for small numbers of study subjects and for the selective nature of the patients studied. Recently, the Intergroup Melanoma Surgi-

Recommended excision margins for invasive primary melanoma	
Thickness of primary	Excision margin
< 1 mm	1 cm
1-4 mm	2 cm
> 4 mm	2-3 cm

cal Trial was published. This study randomized 740 patients with intermediate thickness (1 mm to 4 mm) melanoma and a clinically negative nodal basin to ELND versus observation. They found no overall survival advantage for ELND at a median 7.4 year follow-up. However, there was a small survival advantage described for subgroups of patients who were either less than 60 years of age or had a primary melanoma between 1 mm to 2 mm in depth.¹⁷

Lymphatic mapping with sentinel lymph node biopsy is likely to solve the dilemma of whether or not to perform ELND. With this technique, the surgeon is able to identify the status of a regional nodal basin without doing a full dissection. The biggest advantage of sentinel lymph node biopsy is the ability to accurately identify node-positive patients for further surgical and adjuvant therapy. Node-negative patients could be spared the morbidity of a lymphadenectomy. The concept behind lymphatic mapping and sentinel lymph node biopsy is that melanoma metastasizes in an orderly fashion. Melanoma travels from a primary site through dermal and subcutaneous lymphatic channels to a specific and identifiable lymph node in the regional nodal basin. This will be the first lymph node to receive melanoma metastases. If this lymph node is free of metastases, the other lymph nodes will be negative as well.

Two techniques have been used for sentinel lymph node biopsy. One is an intradermal injection of blue dye made surrounding the primary site. With this technique, patients usually have a preoperative lymphoscintigraphy to verify the correct draining nodal basin. The lymphoscintigraphy is particularly important for patients with trunkal or head and neck lesions, where the lymphatic drainage may be unpredictable. The dye, isosulfan blue (lymphazurin 1%), travels rapidly through dermal and subcutaneous lymphatics. A lymphadenectomy incision is made over the regional nodal basin. The surgeon dissects the tissue to find a tiny blue lymphatic and follows this blue lymphatic channel to the first draining lymph node.¹⁸ This lymph node is resected for pathologic study.

The other technique is the intradermal injection of technetium-99m sulfur colloid (0.5 - 1.0 mCi), a radioactive substance, immediately adjacent to the primary lesion. A lymphoscintigraphy is performed to confirm the nodal basin draining the primary lesion and to provide a first approximation of the site of the sentinel lymph node. The sentinel lymph node is localized intraoperatively using a gamma probe before the incision is made.¹⁹ This allows a very small incision to be made directly over the sentinel lymph node. The advantages of the technetium sulfur colloid technique is

that 1) it is faster intraoperatively; 2) the correct lymph node can be localized before making the incision, therefore allowing a smaller incision and allowing the procedure to be done under local anesthesia; and 3) it allows intraoperative verification that the node removed is truly the sentinel lymph node.

Early published results of sentinel lymph node biopsy are promising. Donald Morton's group reported 223 patients who underwent intraoperative lymphatic mapping using the blue dye technique.¹⁸ Even early on in their experience they were able to identify a sentinel lymph node in 82% of the patients. The yield has subsequently been higher as the group has gained experience with the technique. They dissected all the regional lymph node basins indicated by lymphoscintigraphy and reported a less than 1% false negative rate.

The technique of intraoperative localization of the sentinel lymph node using a hand-held gamma probe was developed by David Krag at the University of Vermont. Dr. Krag recently reported a series of 121 patients using the technetium sulfur colloid technique with selective dissection of the lymph node basin only for patients with a positive lymph node. They reported that they were able to identify a sentinel lymph node using this technique in 98% of their patients. In 44 patients, they used blue dye as well as the radiocolloid. In this group, all blue sentinel nodes were also radiolabelled. In four patients, a radiolabelled node was detected that had no detectable blue dye. This suggests that the radiolabelling technique is at least as accurate as the blue dye method.²⁰

Sentinel lymph node biopsy is clearly a very simple and accurate procedure. The false negative rate reported is low. There is a very low morbidity associated with this technique. Yet, many questions remain. It has not yet been demonstrated that the routine use of sentinel lymph node biopsy will be associated with improved survival. There is now an ongoing study coordinated by Dr. Morton (the Multicenter Selective Lymphadenectomy Trial) to answer this question. Patients with melanoma >1 mm in depth are being randomized to sentinel lymph node biopsy with subsequent selective lymphadenectomy (for patients found to have metastases in the sentinel lymph node) versus simple observation of the nodal basin. Another issue is whether all newly diagnosed invasive melanoma patients should have a sentinel lymph node biopsy. It is currently not known whether there is a melanoma thickness below which or above which the procedure offers no benefit. It is also not known whether sentinel lymph node biopsy is equally accurate when done before or after wide local excision of the primary. Intuitively, it seems preferable to do the procedure whenever possible before the

wide local excision, as any wide local excision will certainly disrupt lymphatic pathways.

There is now further reason to determine the status of the regional nodes. Alpha interferon has now been approved as an effective adjuvant therapy for resected melanoma patients at high risk of systemic relapse. ECOG Trial EST 1684 randomized patients at high risk of systemic relapse (resected node-positive patients or those with primary lesions >4mm) to a year of high-dose interferon alfa-2b versus observation. At a median follow-up time of 6.9 years, the group that received interferon had a significant improvement in disease-free and overall survival. Interferon alfa-2b is the first adjuvant demonstrated to show a significant benefit for melanoma patients at high risk of systemic relapse.²¹

Conclusions

The appropriate excision margins for primary melanoma are well established. Wide local excision in most circumstances can be done without complex flaps or skin grafting. Local recurrence rates should be less than 5%. The management of the regional nodal basin is still somewhat controversial. With increased acceptance of sentinel lymph node biopsy, the controversy regarding ELND should pass. When we counsel patients with newly diagnosed invasive melanoma regarding management of the regional lymph nodes, we discuss options for ELND, observation, and sentinel lymph node biopsy. We offer sentinel lymph node biopsy fairly freely, and it has met with high patient acceptance. To optimize the accuracy of the sentinel lymph node biopsy, we recommend decisions regarding the management of the regional nodal basin take place before the patient undergoes a wide local excision of the primary.

The keys to lowering melanoma mortality lie in prevention and early diagnosis. When melanoma is diagnosed early, it is usually curable with surgery alone. Patients with *in situ* melanomas have a five-year survival rate near 100%. Patients with thin melanomas (less than 1 mm) have a five-year survival of 90% or greater. We also advocate a comprehensive, multidisciplinary approach to melanoma. Detailed dermatologic screening for early lesions, accurate pathologic review, prompt and accurate surgical management and nodal staging, and adjuvant therapy, when indicated, will all contribute to the best patient outcomes possible.

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Uncommon cutaneous neoplasms

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Introduction

Basal cell carcinoma, squamous cell carcinoma, and melanoma are the most common cutaneous neoplasms. The importance of disseminating information regarding these malignancies is readily apparent. There are, however, other important types of skin cancer which are less common but can be encountered by any physician who evaluates cutaneous lesions. Characteristics and therapeutic suggestions for these rare subtypes are presented. They are remarkable for their potential for locally aggressive behavior.

Dermatofibrosarcoma protuberans

Dermatofibrosarcoma protuberans (DFSP) is a very uncommon nodular tumor that originates in the dermis. This low grade sarcoma is characterized by a slow infiltrative growth pattern, which tends to be locally destructive and only rarely metastasizes. Most observers regard this tumor to have a fibro-histiocytic origin.¹ The tumor cells have been shown to exhibit both fibroblastic and histiocytic properties.

DFSPs typically appear in the second to fifth decade. The classic DFSP is most often found on the trunk or the proximal extremities, but face and scalp lesions are also seen. It initially appears as a firm, indurated plaque that may be skin-colored, pink, or hyperpigmented. At this early stage, it can easily be mistaken for a hypertrophic scar or keloid. As the tumor continues to grow, it invades local tissues and becomes multinodular. Lesions are fixed to overlying skin but are moveable over the deeper tissue. It is this stage where the diagnosis is most often made. DFSPs are usually asymptomatic, although larger ones may become painful and ulcerate.



Figure 1. Large DFSP on the right breast, recurrent at the margins of a skin graft.

The tumors exhibit an infiltrating growth pattern. Long-standing lesions can invade fascia, muscle, and bone. In addition, they often grow with pseudopod-like extensions underneath clinically normal skin. It is this subtle growth pattern that helps explain recurrence rates as high as 60% with standard excision² and 20% with wide excision with margins of more than 4 cm (**Figure 1**).³

The metastatic potential for DFSPs is extremely low with estimates of a 1% chance of spread to regional lymph nodes and a 4 % chance of distant metastasis.⁴ Metastatic disease was invariably found in patients who had a history of multiple recurrences. The most common site of distant metastatic disease was the lung.⁴

The diagnosis of a DFSP is easily obtained with a skin biopsy. Incisional or punch biopsies are recommended. The histopathologic diagnosis can be made with great certainty on routine tissue processing, especially in the hands of a dermatopathologist.

Historically, surgical excision has been the primary modality for the treatment of DFSPs. The relentless and

insidious infiltration that characterizes these tumors has led to unacceptably high recurrence rates with standard excisions. Cure rates have been improved with the adoption of wide local excisions, yet average recurrence rates of 20% are still observed. The treatment of choice has recently been established to be Mohs micrographic surgery, which provides very precise and comprehensive margin control. A recent review of the world literature has reported an average recurrence rate of 0.6%.³ The Mohs technique also has the advantage of being tissue-sparing, which is especially critical for facial tumors.

Radiation therapy has played a limited role in the treatment of DFSPs. Recent evidence has been reported supporting its use in the management of residual DFSPs. The role of chemotherapy has been restricted to the treatment of metastatic disease.⁴

Extramammary Paget's disease

Extramammary Paget's disease (EMPD) is an uncommon apocrine gland tumor which most commonly appears in the female perineum. It is characterized by slow growth and eczematous appearance.

Three popular theories explain the pathogenesis.^{6,7} The first is that the tumor cells in the epidermis represent an *in situ* adenocarcinoma of the epidermis with tumor cells migrating along apocrine and eccrine ducts to the epidermis. The second theory is that of extension to the epidermis (epidermotropic spread) from an underlying adenocarcinoma of the gastrointestinal or genitourinary tract. The third theory, which is the most popular, is based on an adenocarcinoma developing in a pluripotent cell in the epidermis.

The classic picture of extramammary Paget's disease is a slowly enlarging pink-to-red flat plaque. Epidermal changes of hyperkeratosis, crusting, and superficial ulceration are common. Eighty percent of the patients in one study had vulvar lesions.⁸ Other sites include scrotum, axilla, perianal area, groin, and buttocks. Pruritus, or a burning sensation, are frequent complaints. The tumor is most often confused with a superficial fungal infection, a nonspecific dermatitis, or Bowen's disease. A delay in diagnosis averaging nearly two years is common. Diagnosis is confirmed by punch biopsy.

EMPD usually does not invade deeply into the dermis. The most remarkable feature of this tumor is the tendency for lateral extension into clinically normal epidermis. This extensive subclinical involvement of skin by tumor explains why recurrence rates range from 12% to 60 % after excisional surgery.^{9,10,11,12}

Unlike mammary Paget's disease, which is a skin extension of an underlying adenocarcinoma of the breast, only on rare occasions do patients with extramammary Paget's disease have an underlying intraductal adenocarcinoma. Patients with EMPD do have an increased risk of up to 12% of having an associated visceral neoplasm.¹³ In these uncommon situations, the underlying tumor correlates with the site of the cutaneous involvement, so that vulvar and scrotal lesions are associated with genitourinary tract malignancies, and rectal lesions are associated with gastrointestinal adenocarcinoma.

The first step in the management of patients with EMPD is to rule out the presence of an associated visceral malignancy. Once this is complete, surgical removal of the tumor can be performed. Conventional surgical excision is not recommended because of the high recurrence rates. Various efforts have been made to increase surgical cure rates and include multiple satellite biopsies,¹⁴ Mohs micrographic surgery,^{14,15,16} and traditional frozen sections.¹⁷ Some type of frozen section control of the surgical margins does provide the patient with higher reported cure rates.

Verrucous carcinoma

Verrucous carcinoma (VC) is a rare squamous cell carcinoma characterized by a slow-growing, warty tumor with a low propensity for metastases. It is most often found on the penis, foot, and the oral cavity. Histologically, this squamous cell carcinoma is noted for its high degree of differentiation without cellular atypia.^{18,19,27} Human papillomavirus infection has been linked with some anogenital VC. Tobacco has been linked with VC of the oral cavity.^{19,20}

These neoplasms initially appear like a wart, demonstrating a verrucous, exophytic quality. With time they can invade deeply into subcutaneous tissue. There are three major forms of VC.

Plantar verrucous carcinoma is a variant confused with a recalcitrant wart. It is most often seen on the soles and predominantly in men.²¹ As the tumor grows, it frequently develops crypts filled with hyperkeratotic material. Without treatment, VC can penetrate plantar fascia and may even destroy bone.

Verrucous carcinoma of the oral cavity presents as a confluent mass of whitish, cauliflower-like lesions on the buccal mucosa. Not uncommonly, secondary infection occurs within these lesions, leading to regional adenopathy which can be mistaken for metastases. Tumor spread to regional lymph nodes rarely occurs, and distant metastasis is even less common.



Figure 2. Verrucous carcinoma of the penis.

Anogenital verrucous carcinoma is the third variant and is most commonly found on the penis, where it appears as a papillomatous hyperkeratotic lesion (Figure 2). If allowed to grow, this tumor can infiltrate deeply, destroying the glans or penile shaft. This variant also occurs on the vulva or perianal skin.

VC is a slow-growing neoplasm. Delays in diagnosis are commonplace, which means this relentless tumor often has the opportunity to produce significant local soft tissue or bone destruction. Because metastatic spread is uncommon, the associated mortality is low.

A clinical and microscopic correlation is required to best diagnosis VC. The presentation of a large, intractable wart-like lesion is often the first clue that further investigation is warranted. A deep biopsy is required to observe the very well-differentiated histologic features of the squamous cell carcinoma (SCC). A superficial biopsy frequently reveals only nondiagnostic keratotic changes. The differential diagnosis should include warts, and other forms of reactive epidermal hyperplasias must also be

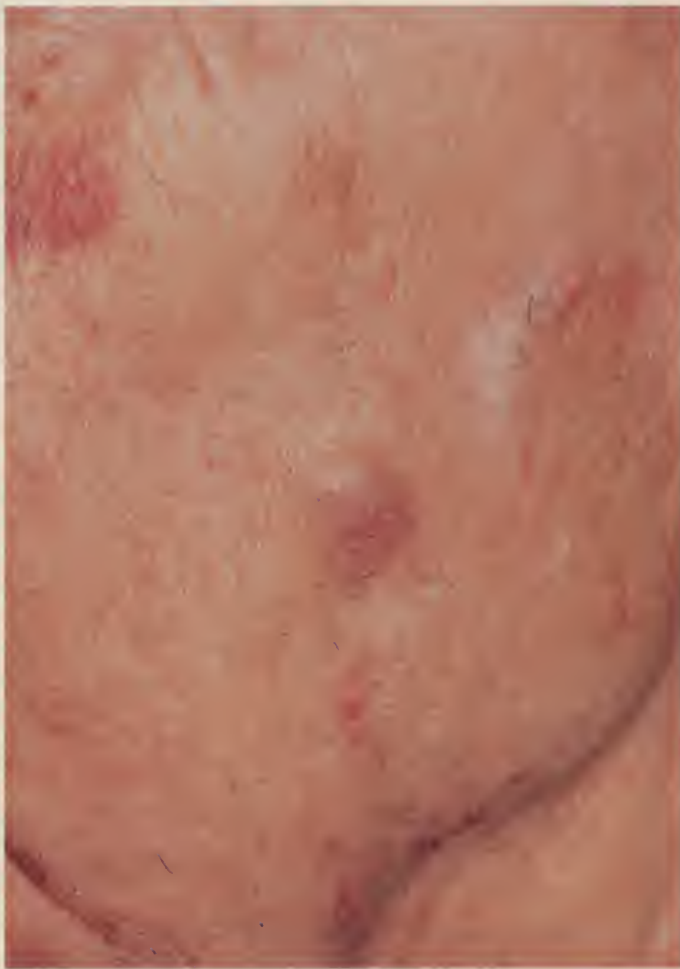


Figure 3. Merkel cell carcinoma on the cheek.

considered, such as deep fungal infections, iododerma, giant seborrheic keratosis, verrucous melanoma, and giant keratoacanthoma.

Surgery is the ideal approach to the management of verrucous carcinoma. A microscopically controlled approach is preferred because of the higher cure rate and the tissue-sparing nature.^{22,23} The role of radiation remains controversial.^{24,25} Reports of anaplastic transformation have been described after this treatment modality.²⁶

Merkel cell carcinoma

Merkel cell carcinoma (MCC) is an unusual and highly malignant tumor of the skin which may act similar to melanoma.²⁸ The typical nodule is solitary, deep red-to-purple, and dome-shaped appearing on older Caucasians (**Figure 3**). They may ulcerate if not excised quickly enough. Half the lesions are found on the head and neck, another 40% on the extremities. It is frequently misdiagnosed clinically as a basal cell carcinoma, melanoma, Kaposi's sarcoma, or pyogenic granuloma. Excisional

biopsy is recommended, since diagnostic criteria may be better evaluated with substantial tissue sampling. The tumor takes its name from its neuroendocrine-presumed cell of origin, the Merkel cell. This cell plays an unclear role as part of the nerve complex responsible for touch transmission.^{28,30}

MCC follows a course somewhat parallel to that of melanoma. Patients are often treated accordingly. Following a chest x-ray and thorough history and physical exam, a wide excision of the lesion should be undertaken with margins recommended at 2.5 cm to 3.0 cm. Mohs micrographic surgery has been used and reported, but its use is not widespread. Sentinel node biopsy may be useful to determine nodal involvement. One recent study demonstrated a significant improvement in outcome when complete excision was followed by adjuvant radiation therapy.²⁹ Metastatic disease is sometimes addressed with chemotherapeutic agents similar to those used for small cell carcinoma of the lung.³¹

Prognosis in MCC is relatively unfavorable. It is estimated that a third of patients will die from their disease, although no large five-year survival rates are published.^{29,30}

Atypical fibroxanthoma

Atypical fibroxanthoma (AFX) is another uncommon cutaneous malignancy with a generally less aggressive profile. Enziger and Weiss feel it is a low grade variant of malignant fibrous histiocytoma.³² It has been described as "bizarre, cytologically malignant but usually clinically benign."³³ Most arise as pink, translucent, ill-defined papules or nodules on actinically damaged skin of the head and neck in the elderly. They may be crusty or ulcerate.³⁴ Frequently, their rapid growth often initiates the patient's visit. Since they mimic more common cancers, the diagnosis is rarely made clinically. Usually the biopsy results surprise the clinician. Similar to squamous cell carcinoma, immunosuppression and a history of ionizing radiation are other predisposing factors.

The spindle cell histology often causes confusion with other spindle cell variants, such as spindle cell squamous cell carcinoma, desmoplastic melanoma, or dermatofibrosarcoma protuberans. Immunohistochemical stains are useful in sorting out these microscopic differences.³⁵ While excision is the treatment of choice, indistinct clinical margins and frequent recurrences dictate the usefulness for margin control with Mohs surgery.³⁶

Microcystic adnexal carcinoma

Microcystic adnexal carcinoma (MAC) is another uncommon cancer of the face, often in the perioral skin. Because of its location on sun-damaged skin, there is a common assumption that ultraviolet radiation plays a part in its development. It often has a benign appearance, rarely ulcerating or bleeding. The lesion may appear as an indentation of minimally sclerotic skin with or without subtle changes in sensitivity.³⁷

The diagnosis may be confirmed with a biopsy. However, attention should be paid to biopsy method since a superficial one (e.g., shave biopsy) might be too superficial or too insignificant to reveal all the characteristic histology.³⁸ From its immunohistochemical staining properties, MAC shares histogenic features of both follicular and sweat gland origins.³⁹

Although it resides in the dermis, very little change is seen in the overlying epidermis. Indeed, its *sine quo non* is a histologic margin far broader than the clinical ones (Figures 4 and 5). When removed using simple excision techniques, there is a frequent finding of positive tumor margins requiring a return to the operating room. This has led a number of observers to name this an "aggressive" tumor, when in fact, there is no evidence of rapid growth. A better term would be "covert," since the true size of the cancer is simply hidden. Nevertheless, perineural invasion is frequent, and it can occasionally invade underlying muscles. In advanced lesions, there is stromal sclerosis of the invaded



Figure 4. Clinically small microcystic adnexal carcinoma of the upper lip.



Figure 5. Same lesion following excision using Mohs micrographic surgery. Wound size represents tumor size and an additional 1mm to 2 mm margin.

deep dermis, sometimes rendering the dimpled or retracted appearance clinically.³⁹

There is a high frequency of incomplete excision with subsequent local recurrence. This can be expected from the clinically unrecognizable extension of the tumor. Therefore, Mohs micrographic surgery is particularly well-suited for treatment.^{37, 38}



Figure 6. Classic Kaposi's sarcoma.

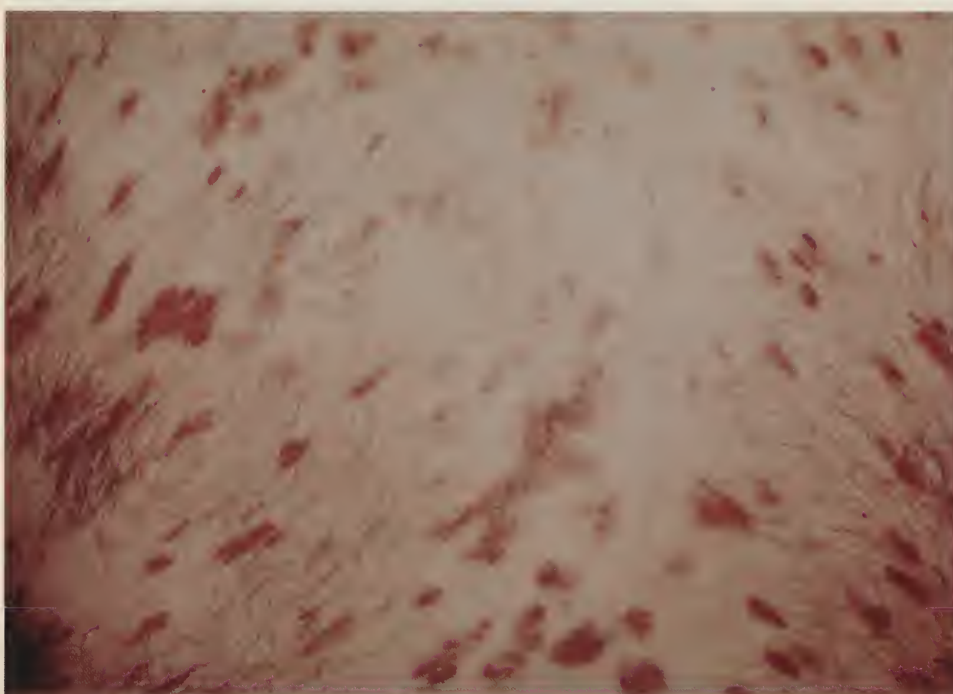


Figure 7. Disseminated KS on the back of an immunocompromised patient. Note the pattern of lesions falling within the natural skin lines.

Kaposi's sarcoma

Three clinical settings delineate separate presentations of this tumor. The traditional rare Kaposi's sarcoma (KS) was seen on the distal lower extremity of Ashkenazic Jewish men and Mediterranean phenotypes. A second endemic of this tumor was seen in Africa, particularly in equatorial countries. Over the last two decades, there has been a remarkable increase in the

incidence of this tumor in a third clinical setting, that of immunosuppression in general and HIV infection in particular.^{40,48}

Early, the lesions appear as flat, red or violaceous dermal macules or papules. As they progress, the lesions become multinodular plaques and deep purple in color. The distribution can be widespread and generalized in the immunosuppressed. In classic KS, lesions are confined to the lower extremity, particularly the distal skin of the ankles and feet (Figure 6). These patients may have concomitant gastrointestinal involvement, but this is often an autopsy finding rather than one of clinical importance.⁴¹ HIV-associated KS tends to follow a distinct course, one more relentless and widespread than the other types. In contradistinction to classic KS, HIV patients develop numerous tumors of the face, oral mucosa and trunk, often following cleavage lines, and have fewer distal extremity lesions (Figure 7). Gastrointestinal⁴² and pulmonary⁴³ involvement can be devastating.

The association of KS with another virally-induced epidemic, coupled with the recent ability to search for biochemical evidence of viral invasion, prompted the subsequent discovery of the "Kaposi's sarcoma-associated herpes virus (KSHV)." Dictor, et al. recently confirmed the close association of KSHV in classic and HIV-associated KS.⁴⁴

Treatment must be tailored to the patient. With fewer lesions and a more protracted course, classic KS can be addressed with ionizing radiation therapy⁴⁵ or intralesional chemotherapy.⁴⁶ In widespread or life-threatening dis-

ease sometimes seen in the later stages of AIDS, more aggressive systemic chemotherapy has been palliative and useful. Combination adriamycin/bleomycin/vincristine is slowly being replaced by the newer liposomal doxorubicin (Doxil) which appears to be as effective but better tolerated.⁴⁷ The recent experience with triple regimen therapy (protease inhibitors, nucleoside, and non-nucleoside reverse transcriptase inhibitors) for HIV has shown dramatic promise for both AIDS and the associated KS. Since experience plays a great role in handling this newer, but now common epidemic, appropriate patient referral may be the key to successful therapy.

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Cosmopolitan, April 1997

The CMERC Update, now in its second year, informs all Med Chi accredited CME sponsors about the activities of the Continuing Medical Education Review Committee (CMERC). The CMERC has received feedback (both negative and positive) from our accredited sponsors this past year. This exchange of information has kept us all better informed.

New guidelines and policies on mergers, acquisitions, and organizational restructuring

Many Med Chi accredited sponsors have been involved in mergers, acquisitions, and organizational restructuring this year. Additionally, the Accreditation Council for Continuing Medical Education (ACCME) recently developed *A Policy to Handle Continuation of Accreditation After Corporate Restructuring*. These events prompted the CMERC to establish a task force to develop our own policy covering these issues. At the March CMERC meeting, the *Guidelines and Policies on Mergers, Acquisitions, or Other Organizational Restructuring Involving CME Accredited Institutions* were approved. It is hoped that the document will be reviewed and approved by the Med Chi Board of Trustees at their April meeting.

Guidelines and standards used for CME program accreditation

A question was raised regarding the essentials, standards, and guidelines used by the CMERC in making accreditation decisions. In addressing this issue, it is important to remember that the CMERC was formed around 1969 to 1970, about a decade before the ACCME was formed. The CMERC started to conduct CME surveys and accredit CME sponsors using guidelines and standards derived from the American Medical Association (AMA). Accredited CME sponsors were also required to report to the AMA annually.

In 1981, the ACCME was formed and they developed and approved the seven *Essentials for Accreditation* in 1983. Subsequently, they began the process of accrediting state medical societies as intrastate accrediting bodies for CME sponsors. This was done through CRR (Committee

for Review and Recognition) surveys in those states that require continuing medical education.

Intrastate accrediting bodies have always been required to have their own essentials and standards for accreditation, which must be consistent with the ACCME's essentials. They can be more, but may not be less, stringent than the ACCME's essentials. The CMERC has adopted essentials and standards that are identical to the ACCME's.

CME programs surveyed for reaccreditation (April 1996 - April 1997)

Sixteen CME programs were surveyed by the CMERC. Of the 16, nine were reaccredited for four years (the maximum currently allowed). One program was reaccredited for three years; five were reaccredited for two years (including one program that was resurveyed after probation, when two years is the maximum that can be granted); and one was reaccredited for one year.

Of the nine programs that were reaccredited for four years, four did not have any problems with compliance with any of the seven essentials. One sponsor had only one problem (with Essential #6). This is very encouraging; last year, only two programs were without compliance problems.

The CME chairs of two of the programs spoke at the CMERC workshop at the Med Chi annual meeting. They were Clifford G. Andrew, M.D., Anne Arundel Medical Center, and Richard A. Farson, M.D., Southern Maryland Hospital.

Among the twelve CME sponsors surveyed, the most frequently found problems related to Essentials #2, 3, 4, and 5. Overall, the results show many CME programs doing much better; only three had serious problems with compliance.

Interim report reviews completed (April 1996 - April 1997)

Twenty-three interim reports were reviewed, and all but two were found to be acceptable. One sponsor that was asked to submit another interim report was placed on probation for failure to do so. Another sponsor was placed on probation due to failure to correct previous problems. One progress report was reviewed and accepted.

Consultations performed (April 1996 - April 1997)

Nine consultations were held. Most of the consultations were initiated by the CMERC when problems that could jeopardize continued accreditation were identified.

News from the ACCME

The proposed changes to Essential #7 have been ratified by the members of the council. However, the revised version will not be final until the seven parent organiza-

tions of the ACCME take action. The final action will probably occur in June; we will let you know. (The parent organizations of the ACCME are: the American Medical Association, the American Hospital Association, the American Board of Medical Specialties, the Association of American Medical Colleges, the Association of Hospital Medical Education, the Council on Medical Specialty Societies, and the Federation of State Medical Boards.)

DEUSDEDIT L. JOLBITADO, M.D.

Dr. Jolbitado is a member of Med Chi's Continuing Medical Education Review Committee ■

The purpose of this column is to inform CME chairpersons, CME committee members, and all interested physicians about the activities of Med Chi's Continuing Medical Education Review Committee (CMERC) and about rules and procedures that affect the implementation of CME programs in Maryland. Where appropriate, news from the Accreditation Council for Continuing Medical Education (ACCME) will be included.

Med Chi Bicentennial Celebrations

*Med Chi has already begun planning celebration activities
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Morningstar risk-adjusted performance ratings for the periods ended 2/28/97

Fund	Overall	3 yr	5 yr	10 yr
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Blue Chip Growth	★★★★★	★★★★★	—	—
Capital Appreciation	★★★★★	★★★★	★★★★	★★★★★
Dividend Growth	★★★★★	★★★★★	—	—
Equity Income	★★★★★	★★★★★	★★★★★	★★★★★
Equity Index	★★★★★	★★★★★	★★★★★	—
Growth & Income	★★★★★	★★★★★	★★★★★	★★★★
Growth Stock	★★★★	★★★★	★★★★	★★★
Mid-Cap Growth	★★★★	★★★★	—	—
New America Growth	★★★	★★★	★★★	★★★
New Era	★★★★	★★★★	★★★★	★★★
New Horizons ⁺⁺	★★★	★★★	★★★	★★★
OTC ⁺	★★★	★★★★	★★★	★★
Science & Technology	★★★★	★★★	★★★★	—
Small-Cap Value ⁺⁺	★★★★★	★★★★	★★★★★	—
Spectrum Growth	★★★★	★★★★	★★★★	—

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†Performance prior to 9/1/92 reflects investment managers other than T. Rowe Price. ††These funds are closed indefinitely to new investors.

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May/June, 1997

Selected Communicable Diseases in Maryland in 1996

This report, the first of a three-part series to be continued in July and August, describes the epidemiology of selected communicable diseases reported to the Epidemiology and Disease Control Program in 1996. Communicable disease reporting is mandated by both Maryland law and regulation. This passive system receives reports primarily from health care providers and laboratories; some reports come from other sources. The communicable disease staff at the State's 24 local health departments verify that cases meet a standard clinical and/or laboratory case definition of the Centers for Disease Control and Prevention (CDC). Local investigators may also seek to identify risk factors for disease acquisition, provide patient education to reduce the spread of disease, identify contacts for prophylactic treatment, and enact other public health control measures when appropriate.

Each week data from the 24 local health departments is combined at the Division of Communicable Disease Surveillance at DHMH and then transmitted to the CDC for Morbidity and Mortality Weekly Report (*MMWR*). These provisional weekly data are then further reviewed, edited, and analyzed at the local and state health departments to produce this yearly summary.

In this report the number in parenthesis after the title of each disease indicates the number of cases reported in 1996 in Maryland. (Through 1993, this yearly report summarized cases with onset in the reporting year.) Below the disease name is the

incidence rate for Maryland and the preliminary rate for the United States. Because final national figures are not published until later in the year, the number of U.S. cases is taken from the cumulative number of cases reported in *MMWR* for 1996 (CDC, *MMWR*, January 3, 1997, Vol. 45, Nos. 51 & 52). All incidence rates throughout this report are given as cases per 100,000 population. The population figures used in rate calculations come from the Maryland Office of Planning and the U.S. Census Bureau (July 1, 1996 Population Estimate).

The prompt, accurate, and complete reporting by physicians, other health care providers, laboratories, etc., is extremely important to achieving our goal to describe and control communicable diseases in Maryland. We are committed to seeking innovative means to facilitate physician reporting, to establishing electronic laboratory reporting to increase timeliness and completeness, and to providing improved accessibility to surveillance data via our internet home page (www.charm.net/~epi1). We gratefully acknowledge the contributions of local health departments, infection control professionals, laboratories, physicians and other health care providers who provided essential information. Thanks are also due the DHMH staff who assisted in case investigation and data analysis, and to Ms. Anne Jones, who coordinated the data entry and editing.

BOTULISM (1)

0.02/100,000 (U.S. 0.05/100,000)

Clostridium botulinum type B toxin was isolated from the stool of a 10 week old infant who had a weak cry, constipation, muscle weakness, and respiratory difficulties. Symptoms began several days before hospital admission. The infant recovered with supportive care, and without sequelae. The source of the infection was not identified. The infant was both breast fed and given formula.

HAEMOPHILUS INFLUENZAE DISEASE (76)

1.5/100,000 (U.S. 0.4/100,000)

The 76 reported cases of invasive disease due to *Haemophilus influenzae* (all serotypes and among all ages) included bacteremia (51 cases, 67%), pneumonia (12 cases, 16%), meningitis (11 cases, 15%), and other infections (2 cases, 3%). Figure 1 illustrates the trend for invasive *Haemophilus influenzae* disease (all serotypes) for the past 11 years (1986-1996). Active surveillance for invasive *H. influenzae* disease of all serotypes and among all ages began in November 1991.



Figure 1. *Haemophilus influenzae* disease (all serotypes). Incidence in Maryland, 1986 - 1996.

Table 1a and Figure 2 show the number of cases by jurisdiction.

The overall incidence rate for males was 1.3 (32 cases) and 1.7 (44 cases) for females. The rate for whites in 1996 was 1.3, and 2.0 for non-whites. The case fatality rate for known outcomes in 1996 was 9.2%, which is a decrease from 1995 (14.9%).

Cases ranged from one day old to 97 years (median 41 years). The number of cases by age category from 1987 to 1996 is shown in Figure 3. The number of cases in children 2 months to 5 years of age, the vaccine preventable age category, was 13. However, only three of 13 were cases of *H. influenzae* serotype b, which is the same as in 1995.

Data on the serotypes of *H. influenzae* isolates were available for 63 (83%) cases. The breakdown by serotype for these cases is: 32% serotype b, 41% nontypeable, and 27% other (71% of these others were type f).

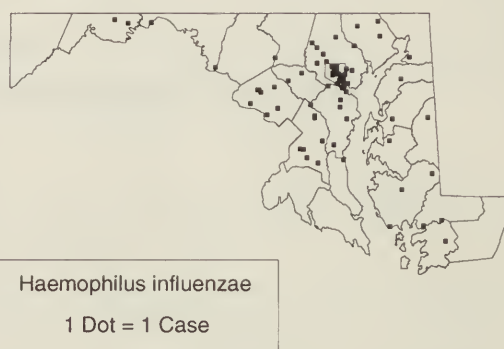


Figure 2. *Haemophilus influenzae* invasive disease (all serotypes), Maryland, 1996.

Only 3 of the 22 *H. influenzae* type b disease (Hib) cases were the appropriate age for Hib immunization. A 9 month old had been vaccinated against Hib, but the dates of vaccination are not known; a 6 month old child was reported to be unvaccinated against Hib; and the vaccine status of a three month old was unknown. The other cases of type b disease were 10 years of age and older.

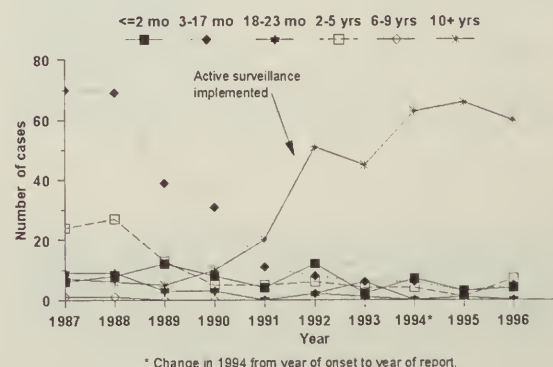



Figure 3. *Haemophilus influenzae* invasive disease (all serotypes). Cases reported by age group, Maryland, 1987 - 1996.

Table 1a. Cases of Selected Notifiable Diseases Reported in Maryland in 1996 by County

<div><div>DMH</div><div>Maryland Department of Health and Mental Hygiene</div></div>			Jurisdiction	Population	Infectious Parotitis (Mumps)	Pertussis	Rubella (German Measles)	Rubeola (Measles)	Hepatitis A	Hepatitis B	Hepatitis NANB	Encephalitis	Haemophilus influenzae Disease	Meningococcal Disease	Meningitis, Bacterial	Meningitis, Aseptic	Salmonellosis	Typhoid Fever	Shigellosis	AIDS	Chlamydia	Gonorrhea	Syphilis, Primary and Secondary	Syphilis, Congenital	Tuberculosis	Lyme Disease	Rocky Mountain Spotted Fever	Animal Bites	Rabies, Animal	
	Allegany	74,924			1	25			2	3		3	2		1	1	10		1	3	69	43			1	1		353	8	
	Anne Arundel	463,199			7	25			18	18	1	4	6	8	8	16	76		72	66	616	334	20	1	13	21	7	1,127	89	
	Baltimore City	691,465			3	42			41	33	1	2	25	13	34	10	286		206	1,224	13,616	6,301	632	34	103	1	1,356	3		
	Baltimore Co.	713,527			3	21		1	33	17	2	2	9	10	11	21	166	3	72	129	942	620	59	4	36	99	2	915	65	
	Calvert	66,203			3	3			5			2			2	7	8		9	4	85	47			2	14		113	17	
	Caroline	29,321				2						2	1	1	1		17		12	2	111	51			5	2	1	117	13	
	Carroll	140,660			2	9			3	2	1			2	1		16		4	9	72	26	1	1	4	27	2	130	26	
	Cecil	79,362			1	2			2	2			1	1		1	9			4	36	22			16			269	6	
	Charles	114,925			1	1		1	5	10					4	4	16		32	18	227	128	5		1	17	2	423	13	
	Dorchester	30,414							4				3		2	1	17		23	6	113	161	5		1	1	2	151	15	
	Frederick	180,204			54				5	4	1	1	1	1	2	5	22		12	9	159	119	1		3	4		293	105	
	Garrett	29,735							1	2					1		1	1		1	15		1					98	10	
	Harford	212,165			2	20			16	7			3	2	3	10	35	2	9	25	258	103	2		7	43		551	15	
	Howard	223,167			1	31			8	2			2		5	8	33	2	10	28	141	80	6		5	28	1	379	24	
	Kent	18,778											1				9		8		33	7			1	22		55	1	
	Montgomery	816,985			3	24			54	21		7	7	2	16	48	156	8	98	189	651	437	11	1	51	59	2	1,350	47	
	Prince George's	778,139			4	12			45	41		2	8	9	17	43	162	1	252	377	2,550	2,039	58	4	62	45	1	1,117	42	
	Queen Anne's	38,114											1	2	1		5		5	4	40	18	1		3	26		136	19	
	Saint Mary's	82,282			6	2			6			1				3	15		6	5	140	74	1		3	6	16	235	7	
	Somerset	24,529				3				1			1			3	21		11	2	111	81			1	1		112	11	
	Talbot	32,759				3				1			1		2	3	6		4	3	88	50	1		3	12	1	123	15	
	Washington	128,126				12			8	5	1	3	2	2	1	23	30		3	11	118	125			3	1		310	19	
	Wicomico	80,232				4					1		2	3	1	3	34	1	122	24	344	356	2		9	1	1	510	46	
	Worcester	40,389				7								3			10		13	8	170	94	2		2	1		192	21	
	Not Stated																			127										
	Maryland Total	5,089,604			37	278	0	2	256	169	8	30	76	58	113	210	1,160	18	984	2,278	20,705	11,316	808	45	319	448	38	10,415	637	
	Maryland Totals for Prior Years	1995 1994 1993 1992 1991			41 65 85 84 236	49 53 167 52 61	1 0 2 5 1	221 198 184 254 178	262 351 288 388 258	6 23 16 31 45	35 33 25 22 27	74 80 64 81 40	42 28 50 31 35	172 175 158 165 166	323 244 257 223 333	1,215 1,167 1,028 1,021 1,262	18 14 8 7 8	984 639 453 430 258	2,278 2,500 1,617 1,273 841	20,705 14,675 16,823 13,832 16,513 21,464	808 492 326 393 590 1,016	45 24 22 36 62 54	319 368 343 417 442 451	448 368 207 185 283	38 36 20 23 16 22	10,415 10,491 10,396 10,349 10,676 9,927				

Table 1b. Cases of Selected Notifiable Diseases Reported in Maryland in 1996 by County

		Jurisdiction	Population	Amebiasis	Anthrax	Botulism	Brucellosis	Campylobacteriosis *	Cholera	Giardiasis *	Kawasaki Syndrome	Legionellosis	Leprosy	Leptospirosis	Listeriosis *	Malaria	Mycobacteriosis, Non-TB	Newborn Septicemia	Plague	Poliomylitis	Psittacosis	S. typhi Carrier	Tetanus	Trichinosis	Tularemia	Typhus, Murine	Vibrio (Non-O1) Infection	Yersiniosis	
		Allegany	74,924	1				13		27	3	4			3	3	2										1	1	
		Anne Arundel	463,199					37		30					4		42	3									1	1	
		Baltimore City	691,465	3				62				3	1		4		203	3									1	7	
		Baltimore Co.	713,527	3							1	8			3	8	74	8											
		Calvert	66,203							2		2					4												
		Caroline	29,321					1																					
		Carroll	140,660					10		5		3					8												
		Cecil	79,362			1				7							6												
		Charles	114,925					4		1	1	2					5												
		Dorchester	30,414									1					2												
		Frederick	180,204					17		12		3					25												
		Garrett	29,735																										
		Harford	212,165					16		8	2	1			1	1	15	4											
		Howard	223,167					9		12	1	1				2	20												
		Kent	18,778														1												
		Montgomery	816,985	2				40		20	5	4			1	32	79	2											
		Prince George's	778,139	5				32		12	15	5			1	40	121	28									2		
		Queen Anne's	38,114					1		1							4									2			
		Saint Mary's	82,282	1				2		3							8												
		Somerset	24,529					5									10												
		Talbot	32,759														1												
		Washington	128,126	1				2		3		2					12												
		Wicomico	80,232					24		3	1					1	47	6											
		Worcester	40,389					7		2							18	3											
		Not Stated															95												
		Maryland Total	5,089,604	16	0	1	0	282	0	148	29	39	0	1	13	87	802	54	0	0	0	0	0	0	0	0	2	4	8
		Maryland Totals	1995	11	0	1	2	206	0	147	39	29	2	1	10	63	1040	93	0	0	2	0	0	0	0	0	1	6	13
		for	1994	12	0	1	1	204	1	108	36	82	0	1	19	83	1048	77	0	1	2	2	1	0	1	1	1	18	
		Prior Years	1993	9	0	1	0	141	0	107	30	52	0	1	9	57	695	133	0	1	1	1	2	0	0	0	1	24	
			1992	10	0	1	0	150	3	69	26	39	0	0	25	61	577	136	0	0	2	4	0	3	0	1	5	23	
			1991	9	0	0	0	119	4	90	17	34	5	0	2	62	507	84	0	1	5	2	1	0	2	0	2	28	

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| 25th annual geriatrics symposium: current topics in geriatrics , sponsored by the Johns Hopkins Geriatrics Center, at the Renaissance Harborplace Hotel, Baltimore. Credits: 19 Cat 1 AMA credits. Fee: \$400/physicians; \$300/residents, fellows, allied health professionals. | Aug. 21-23 |
| Fifth annual progress in hematologic malignancies and bone marrow transplantation , sponsored by the division of hematologic malignancies, department of oncology, Johns Hopkins. Credits: 7.5 Cat 1 AMA credits. Fee: \$100/alumni past registrants; \$125/new registrants. | Sept. 19 |
| 23rd annual topics in gastroenterology and liver disease , sponsored by the Meyerhoff Center for Digestive Disease. Credits: 24 Cat 1 AMA credits. Fee: \$535/physicians; \$285/residents, fellows. If postmarked prior to August 1: \$495/physicians; \$250/residents, fellows. | Sept. 24-26 |

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University of Maryland School of Medicine (continued)

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Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

- | | |
|--|------------------|
| 56th annual American occupational health conference: discover the reality , sponsored by the American College of Occupational and Environmental Medicine (ACOEM) in conjunction with American Occupational Health Conference (AOHC). Orange County Convention Center, Orlando, Florida. 39 concurrent scientific sessions, 42 postgraduate seminars, and 7 two-day training courses. Contact: Kay Cone, ACOEM, 55 W. Seegers Rd., Arlington Heights, IL 60005, 847-228-6850, ext. 152, Fax 847-228-1856. | May 9-16 |
| Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Physician Memorial Hospital at Hamilton Center, Charles County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. | May 13 |
| Gay and lesbian issues in psychiatry and psychotherapy , 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598. | May 14 |
| TraumaCare '97: the 10th annual trauma anesthesia and critical care symposium and world exposition , Baltimore, MD. Info: ITACCS, P.O. Box 4826, Baltimore, MD 21211, Fax 410-235-8084. | May 15-17 |
| Cutaneous melanoma '97: a clinical symposium for primary care practitioners , sponsored by The Skin Cancer Foundation and Memorial Sloan-Kettering Cancer Center, Memorial Sloan-Kettering Cancer Center, New York, NY. Credits: 6.5 Cat 1 AMA credits. Info: Ludmilla Popoff, 212-639-6754. | May 16 |
| Maryland Public Health Association annual meeting — Maryland's vital stats: addressing premature illness and death , at the BWI Airport Marriott. Info: Kathy Marconi, 301-443-6560. | May 16 |
| The American Psychiatric Association's 150th annual meeting , San Diego, CA. Info: APA Division of Public Affairs, 202-682-6220 (e-mail: paffairs@psych.org). | May 17-22 |
| 2nd Annual mammography — practical challenges of the '90's , sponsored by X-Ray Associates of New Mexico, P.C., at The Eldorado Hotel, Sante Fe, New Mexico. Credits: 20 Cat 1 AMA credits. Fee: \$650/physicians; \$450/residents, fellows; \$350/technologists, nurses. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | May 23-26 |
| How to prevent successful lawsuits in mental health , 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598. | May 28 |
| Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Sacred Heart Hospital, Allegany County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. | May 28 |
| Practical approaches to asthma for primary care physicians , sponsored by Blue Cross and Blue Shield, hosted by AAAAI, 8:30 a.m. to 1:30 p.m. at the Renaissance Harborplace Hotel. Credits: 4.5 Cat 1 AMA | May 31 |

Miscellaneous (continued)

- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Fallston General Hospital, Harford County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **June 4**
- Epidemiology & prevention of vaccine-preventable diseases**, a four-part comprehensive course, presented via satellite by the Centers for Disease Control and hosted by the Maryland Department of Health & Mental Hygiene. Fee: none. Info: Sandra Kash, 410-767-6679. **June 5, 12, 19, 26**
- Family interventions in severe mental illness**, 11:30 a.m. to 1:00 p.m. at The Conference Center at Sheppard Pratt, Baltimore. Credits: 1.5 Cat 1 AMA credits. Fee: none. No preregistration needed. Info: 410-938-4598. **June 11**



PHYSICIAN'S RECOGNITION AWARD

This list reflects a compilation of reports for the months of December, 1996 through March, 1997. Reports were delayed due to installation of a new computer system at the AMA; reports will continue on a monthly basis.

Alvin Abrams	Edward W. Ditto	Mohammad Khodabandelou	Natvarlal Rajpara
Mohamad E. Allaf	Virginia A. Dulany	Stanley A. Klatsky	Charles S. Samorodin
Mirza H.A. Baig	Bayani B. Elma	Peter L. Klein	Michael A. Sauri
Arthur Baitch	John D. Foulke	Morton A. Kress	Everett G. Schaner
Amir S. Banisar	Mary L.S. Furth	Marilyn G. Larach	Philip L. Schneider
Kusay Barakat	Wilmer K. Gallagher	David B. Larson	James A. Shaw
Henry C. Barbot	Carol W. Garvey	Albert K. Lee	Robert A. Shaw
Michael Barth	Jasmine C. Gatti	Donald F. Leon	Ronald N. Shore
Jack Baruch	Sheldon Goldgeier	Donald T. Lewers	John P. Smith
John W. Blenko	Michael D. Goodman	Harvey A. Lewis	Clayton W. Straughn
George R. Brennenman	Robert J.S. Guedenet	Daniel I. Loubé	Scott A. Sweeney
Roy M. Brooks	Mark A. Hendrix	Joselito D. Magday	Barry H. Thompson
Steven A. Burka	Glenn R. Hornstein	Monte Philip Makous	Wanda Tvarian
Harold J. Campbell	W.M. Houk	J. Jon C. Mariano	Margaret A. Weiss
Lois A. Carani	Shai Izraeli	David J. McClain	Sandra L. Welner
Donald E. Casey	Fattaneh T. Jabbari	Samir R.G. Neimat	Sharon A. West
Alain G. Champaloux	Kenneth P. Judd	Rolf Nieman	Robert J. Wilensky
Hong-Yu Chen	Alan S. Kaplan	Talal M. Nsouli	Frederick H. Wilhelm
Mary B. Christensen	Gerson N. Kaplan	Patrick I. Okolo	Hans R. Wilhelmsen
Franklyn W. Colligan	Ronald A. Katz	Marilyn D. Perry	Robert P. Younes
Salvador J. Cosimano	Surinder Kaur	Lawrence D. Pinkner	Shaheer Yousaf
Barbara J. Crain	Marshall P. Keys	Irvin P. Pollack	Evelina Yunan
Fredric D. Daniell	Fauzi Khalil	Martin A. Portillo	Ruthann T. Zern
A.K. Helen L. Dichek	A. Victor Khayat	Ronald Prussick	

Miscellaneous (continued)

Uroradiology in Santa Fe '97, sponsored by the American College of Radiology and the Society of Uroradiology, at the Eldorado Hotel, Santa Fe, New Mexico. Credits: Cat 1 AMA credits TBA. Fee: \$595/physicians; \$395/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

June 15-18

Cancer prevention in community practice, sponsored by the Medical and Chirurgical Faculty of Maryland, at Franklin Square Hospital Center, Baltimore County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.

July 24

Imaging in Santa Fe, sponsored by the American Association of Physician Specialists and the International Institute for Continuing Medical Education, at the Eldorado Hotel, Santa Fe, New Mexico. Credits: 25 Cat 1 AMA credits **pending**. Fee: \$625/physicians; \$425/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

July 28-Aug. 1

Self-Directed CME Activities

Maryland physicians' campaign against family violence, module one: domestic violence, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Maryland physicians' campaign against family violence, module two: child maltreatment, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

Fluorescein angiography conference, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00 a.m. - 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.

Sinai Hospital of Baltimore medical grand rounds, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.

ARE YOU PROVIDING PRIMARY CARE?

WHAT IS YOUR PRACTICE'S STANDARD OF CARE FOR:

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- ◆ Colorectal cancer screening
- ◆ Cervical cancer screening?

Does everybody in your practice know these standards of care?

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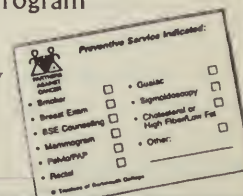
If not 100% (you are not alone), why not?

The Cancer Prevention in Community Practice Program is a user friendly system that helps to answer these questions and improve rates of early detection. We provide assistance to determine standards of care, to assess how well your practice is meeting these standards, and to explore how office flow changes could maximize detection. In addition, we provide manual tools, free for eight months and CMEs/contact hours while you are learning the system.

The Medical and Chirurgical Faculty of Maryland is currently offering this program at the following locations/dates:

- ◆ May 13, 1997 at Physician Memorial Hospital at Hamilton Center, Charles County
- ◆ May 28, 1997 at Sacred Heart Hospital, Allegany County
- ◆ June 4, 1997 at Fallston General Hospital, Harford County
- ◆ July 24, 1997 at Franklin Square Hospital, Baltimore County

Call Carol Schwartz, Project Coordinator at: 1-800-492-1056 or 410-539-0872 to register or inquire further.





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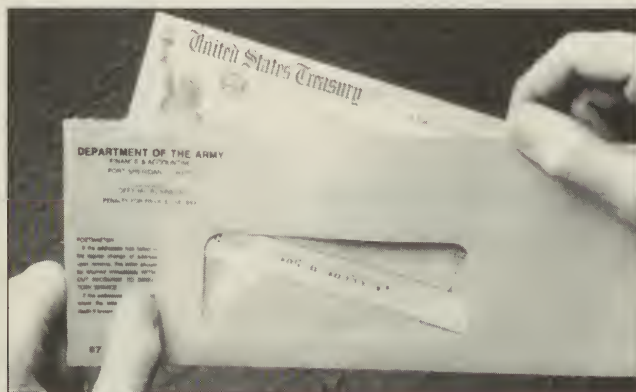


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*For more information, call Vivian Smith at
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PHYSICIAN'S EXAMINING TABLES (2)

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▼ Nonmelanoma skin cancer is much more common in patients who are immunocompromised following organ transplantation. This risk rises progressively with time following the surgery and may be related to the immunosuppressive drugs. Glover, et al. found that Cyclosporine, in particular, is a specific risk factor in the development of squamous cell carcinoma of the skin with an 8.43-fold increase over those transplant patients on other immunosuppressive agents (*Lancet* 1997;349:398).

▼ Ruiz-Maldonado and Orozco recently reviewed "Malignant melanoma in children" (*Arch Dermatol* 1997;133:363-371). The color photographic examples are worth seeking this article, which describes the precursors and congenital abnormalities commonly associated with childhood melanoma. In the same issue, Berg and Lindhof demonstrated that the tremendous acceleration in the incidence of melanoma does not spare the young. Studying nearly 300 pediatric patients in Sweden from 1958 through 1992, they found the incidence of melanoma in adolescents (age 14 to 20) has doubled in the previous decade (*Arch Dermatol* 1997;133:295-297).

▼ Reconstruction of the ala nasi following skin cancer removal is imperative in cases where significant scar retraction can be disfiguring and impair function. Ratner and Skouge describe a simplified technique for "The use of free cartilage grafts in nasal alar reconstruction." Their use of this method as an outpatient procedure under local anesthesia demonstrates its ease, effectiveness, and cost savings over inpatient operating room-based reconstruction (*J Amer Acad Dermatol* 1997;36:622-624).

▼ Dahl, et al. notes cutaneous metastases develop in 2% to 9% of patients with an internal malignancy. Thyroid cancer with skin metastasis is even less common with fewer than 50 patients reported, including the six in their article. In most patients, they found this manifestation one of widely disseminated malignancy and poor prognosis. The average length of survival after diagnosis was 19 months (*J Amer Acad Dermatol* 1997;36:531-537).

▼ Kirsner, et al. report their experience in Miami with "Squamous cell carcinoma arising in osteomyelitis and chronic wounds." Squamous cell carcinoma (SCC) in this scenario is notoriously aggressive with frequent metastasis. In two patients with localized disease, Mohs micrographic surgery (MMS) was curative, while two others with metastatic disease required amputation. Their "series confirms the utility of MMS as a potentially limb-saving procedure in patients with SCC arising in either chronic wounds or in association with osteomyelitis" (*Dermatol Surg* 1996;22:1015-1018).



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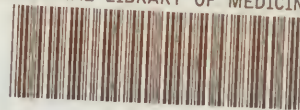
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JULY 1997



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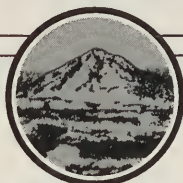


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AVERAGE ANNUAL RETURNS:†

1-year: **4.46%**

Since inception (10/29/93): **3.74%**

Objective: High level of current income

Invests in: Investment-grade corporate bonds

SUMMIT GNMA

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1-year: **5.03%**

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Objective: Capital preservation and liquidity

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Tax-equivalent yield 36% tax rate:

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Current 30-day yield as of 5/11/97:

4.71%

AVERAGE ANNUAL RETURNS:†

1-year: **5.21%**

Since inception (10/29/93): **5.26%**

Objective: High income

Invests in: Investment-grade municipals

SUMMIT MUNICIPAL INCOME

Tax-equivalent yield 36% tax rate:

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Current 30-day yield as of 5/11/97:

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AVERAGE ANNUAL RETURNS:†

1-year: **6.87%**

Since inception (10/29/93): **5.29%**

Objective: Highest possible income

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†Average annual returns are for the periods ended 3/31/97 and include changes in principal value, reinvested dividends, and capital gain distributions. Investment return and principal value will vary, and shares may be worth more or less at redemption than at original purchase.

*Figures are for Taxable Money Market, Short-Term Corporate, GNMA, Municipal Money Market, Municipal Intermediate, and Municipal General Bond Fund categories, respectively, as of 3/31/97. Source: Lipper Analytical Services, Inc.

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Maryland Medical Journal

JULY 1997 • VOL 46 NO 6

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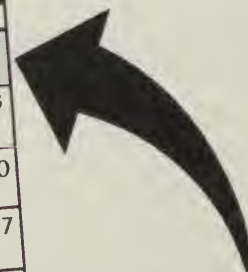
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*Mark Your Calendar
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Preliminary Schedule

Friday, September 5, 1997

- A.M.:** House of Delegates
American Heart Association presentation
Palliative Care
Handling Drug Abusing Patients
- P.M.:** Reference Committees
Telemedicine
Breast Cancer and Prostate Cancer Screening Updates
BPQA Physician Issues

Evening: WELCOME BARBECUE
ON THE BEACH

Saturday, September 6, 1997

A.M.: Exhibits open all day
Plenary Session: Clinical and Practice Tactics
Maryland Society of Anesthesiologists presentation

P.M.: House of Delegates
Risk Management
Internet
Good News about Osteoporosis
Asthma
AIDS

Evening: CRAB FEAST

Sunday, September 7, 1997

A.M.: Plenary Session: Med Chi Credentialing/Managed Care Contracting
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Clarification: A billing service can bill for a percentage of monies



C Sherwood Harris, CCAM, wrote an interesting article titled "Ten rules for the road," (*Md Med J* 1997;46(2):95-96) which I found to be quite good, until I read point number two of his article regarding billing services. Mr. Harris' comment about "it is *illegal* to pay a billing service a percentage based on Medicare collections" is not entirely true. This statement only applies when monies are going directly to the billing agent and deposited to the agent's account, and then the physician is paid. This comes under the issue of "reassignment" and most billing services do not work in that manner.

For clarification on this issue, the Medicare Carrier Manual, Section 3060.10, explains this completely. A billing service can bill a physi-

cian a percentage of monies collected if they are acting as a record-keeping agent only for claims, payment, and billing services. There is no negotiation on their part for payment other than the traditional resubmission of claims for insurance response.

Mr. Harris is absolutely correct when he states that this arrangement must be carefully looked at by the physician who retains a billing service. My recommendation is that this be looked at quarterly though, not yearly.

I wanted to clarify this issue so physicians who use services can be more secure in their decision to do so.

KAREN A. HURLEY, C.M.M., C.P.C.
Hurley Practice Management Services ■

Capitation concept lacks understanding

I read the "SpeakOut" article on capitation by Drs. Mark Seigel and Wayne Spiggle (*Md Med J* 1997;46(1):13) in the January issue. Part of Drs. Seigel and Spiggle's concern really reflects what I believe to be a lack of understanding of the capitation concept. Capitation really represents a budget amount that insurance companies have set aside for delivery of certain types of care. Ultimately, the insurance company is under a global budget based on the income it derives from patient premiums. Within that overall global budget, dollars are set aside for administration, often for an advance for return on investment to its shareholders, and the remainder is based on the company's risk experience into categories for payment of not only physician services, but hospitalization, medication, and diagnostic studies.

Physicians need to understand that neither insurance companies, patients, employers, nor government is going to put more money into the system. Physicians will need to learn how to assess the financial risk of the patient population they are caring for. Capitation represents the dollars to be spent in that patient population, and the more of that financial risk the physician is able to manage effectively, the

more the physician then has control of his medical decision making based on what he or she believes to be in the patient's best interest. Obviously, a healthy patient who is satisfied with the care he or she has received continues to not only participate with that physician, but with that insurance plan, and in that scenario, more members select that insurance plan which therefore has a larger budget, and physicians can then better realize financial independence.

To ignore the faults of the system is to not be completely honest. Insurance companies will spend inordinate amounts for salaries, administration, and set asides for profits. The remedies need to be addressed as to what percentage of the premium is returned to the patient as care, what portion may be set aside for the insurance company to put back into the health care system for those patients with uncompensated care in other areas, and ultimately, the focus needs to be on delivering health care to patients and not return all profits to investors.

MERRILL J. COHEN, M.D., F.A.C.S.,
P.A.
Dr. Cohen practices urology in Greenbelt, Maryland. ■

Reader suggests Poe died from mercury poisoning

What really killed Edgar Allen Poe? It wasn't alcohol; Poe quit drinking heavily six months before his death in the summer of 1849. It wasn't rabies either, despite the interesting presentation of Dr. R.M. Benitez (*Md Med J* 1996;45(9):765-767). The true cause of death may remain a mystery, but I suggest that Poe died of acute and chronic mercury poisoning.

In support of my theory, I am indebted to Poe scholars Jeanne Frank and Samuel Porpora, as well as the research librarian of Med Chi, Russel Kujan.

Following the death of his child bride, Virginia, of tuberculosis in 1847, Poe became a changed man. He became obsessed with the idea that he had or would get cholera. The summer of 1849 was intolerably hot. A cholera epidemic and a malaria epidemic plagued the mid-Atlantic seaboard. Poe had traveled to Richmond from Philadelphia in June 1849. In a letter to his Aunt Maria Clemm dated June 7, 1849, he had begun treating himself with calomel to avoid the fate of thousands of others who were dying of cholera. His calomel use is substantiated by notes from June 1849, when Reverend Charles Burr and attorney George Lippard of Philadelphia both noted Poe's use of calomel.

Calomel, mercurous chloride, was used in Poe's time to prevent cholera, yellow fever, relapsing fever (malaria), bad teeth, and many other problems. The early 1800s was a time of widespread mercury use, especially with physicians such as Dr. Thomas Dover and Dr. Joseph Townsend. Dover, called the quicksilver doctor, was an avid proponent of using sweet salt of mercury (calomel) to treat most all disease, but especially digestive disturbances and emotional problems. It was also known that as little as 6 to 12 grains of calomel could be lethal.

Poe had both emotional and digestive problems. Unfortunately, the more calomel he used, the worse his mental status and digestive regulation became. He used laudanum as well to control his diarrhea, but used ever increasing amounts of calomel.

The malaria outbreak of late summer 1849 cause Poe's ultimate demise. Poe was traveling back to Philadelphia from Richmond with a stop over in Baltimore. He had several previous episodes of delirium with hallucinations, amnesia, sialorrhea, and syncope. His personal physician, Dr. Gibbon Carter, told him that one more attack of Poe's syndrome, which included depression, melancholia, feverish pulse, and collapse, would likely kill him.

Dr. Carter knew that Poe had joined the Sons of Temperance; the "it" that Poe must stop was not alcohol, but it was mercurous chloride, calomel.

In the face of the malaria outbreak, however, Poe increased the dose of his favorite preventive, calomel, which killed him. The description of his death, from Benitez, reflects his acute delirium, with tremors, hallucinations, variable pulse, and sweats. He improved only to suffer oliguria, decreased P.O. intake, anuria, combativeness, followed by coma and death.

Poe had an acute intoxication of mercury which raised his chronic mercury level, causing CNS changes, acute tubular necrosis, acute renal failure, and death.

The additional symptoms that Poe had in his final days of abdominal pain, foaming at the mouth (sialorrhea), headache, amnesia, anorexia, and restlessness are all consistent with chronic calomel poisoning called erethism.

Calomel was widely used as a teething powder as well in Poe's time. Did Poe have bad teeth? At his exhumation in 1879, the undertaker who had buried

Poe said that the body identified as Poe could not be him: the skeleton was too tall and the teeth were too good.

Am I totally certain that Poe killed himself with calomel? I know he had a death wish. I know he had symptoms of a clinical depression. I know that he took calomel regularly. I know that he took extra calomel to prevent cholera and he took a lot of calomel to prevent malaria.

As for my idea of mercury poisoning, Poe said it best: "It is impossible to say how first the idea entered my brain; but once conceived, it haunted me day and night." "Death by Calomel" sounds like a Poe thriller.

RITCHIE C. SHOEMAKER, M.D.

Dr. Shoemaker lives in Pocomoke City, Maryland.

Additional Reading

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- The Pharmacopoeia of the USA. Philadelphia 1842 (How to Make Calomel).
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- Joselow M, et al. Mercurialism: Environmental and Occupational Aspects. *Ann Intern Med* 1972;76:119-130.
- Chang L. Neurotoxic Effects of Mercury: A Review. *Environmental Research* 1977;14:329-373.
- Elhassan S. The Many Faces of Methyl Mercury Poisoning. *J Toxicol Clinical Toxicol* 1982-1983;19(8):875-906. ■

The death of Edgar Allan Poe – a case of syphilis?

I was interested to read the case discussion on Poe. I assume this was based on his original hospital notes. If so, how come they were preserved, and why did they suddenly turn up now? Benitez's diagnosis of rabies¹ brings to mind the aphorism, if you hear hoofbeats, think horses and not zebras, or if attacked, think Spirochetes and not cats.

The former Med Chi president, Sir William Osler, wrote²: "Study one disease, study syphilis thoroughly and you take a knowledge of all others on the way – general medicine, nearly all surgery, and certainly all the specialties." He showed that syphilis was easily the main cause of death, despite having been listed in only tenth place in the UK Registrar General's Report for 1915. Syphilis was "the despair of statisticians." Earlier this century, 67% of Baltimore prostitutes and at least 14% of hospital inpatients in Philadelphia, Boston, and Toronto had syphilis, mostly latent and unsuspected. In a careful autopsy series in Michigan, Warthin found Spirochetes in one-third of these; only a quarter had clinical syphilis.²

I have recently reviewed the literature on musical hallucinations,³ concluding that they arise out of tinnitus in a hyperactive inner ear and underlie creativity in composers and poets, where there is a strong auditory or rhythmic basis to their work. The medical histories of famous composers⁴ and poets strongly support this idea, as most have evidence of hyperactive ears or even the later stages of this syndrome, Meniere's disease. The most striking finding is a very high rate of syphilis, which in its early stages irritates the ear. This eliminates the unlikely scenario that functional psychosis or organic brain disease mediates the often postulated but poorly understood relationship between psychopathology and creativity. Poe is particularly interesting, as he precisely

anticipates this otogenic theory: "True! — nervous — very, very dreadfully nervous I had been and am; but why will you say that I am mad? The disease had sharpened my senses — not destroyed — not dulled them. Above all was the sense of hearing acute. I heard all things in the heaven and in the earth. I heard many things in hell. How then am I mad?" (*The Tell-Tale Heart*).

Pruette⁵ states, "The rumors which still go the round of the clubs in Baltimore claim that Poe was definitely syphilitic." Pruette found this plausible, noting that Poe's character and behavior matched that of other syphilitic geniuses, and that his delusion of persecution in Philadelphia in 1849 was extremely suggestive of the beginnings of paresis. His kindred spirit and great champion, Baudelaire, had syphilis, and like Poe, died in his forties.

I am no expert of syphilis, nor perhaps, are most doctors nowadays. I therefore decided to check if Poe's terminal illness was consistent with syphilis. I did not have far to look, as the first book I picked up⁶ stated that there may be intermittent or fatal fever, especially in chronic alcoholics. Poe had been a notorious drinker, though not apparently

in the six months before his death. His friend Willis⁵ related that with a single glass of wine, Poe's whole nature was reversed, and in the hospital, he vehemently refused to drink alcohol. This strongly implies he had acquired through disease or illness a physiological antipathy to alcohol. Such intolerance of alcohol is characteristic of the hyperactive ear syndrome,³ as it aggravates still further the labyrinthine pressure disturbances at its root.

A.G. GORDON

Dr. Gordon resides in London.

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Author responds

I was intrigued by the theory that *lues venereum* may have played a role in Mr. Poe's death, but I find several discrepancies which I would like to address.

It has been said that "he who knows syphilis, knows medicine," but it should also be remembered that syphilis has often been called "the great imitator" and that it is sometimes difficult to distinguish the imitation from the genuine article. While the clinical manifestations of

syphilis have been known (and the origins argued over) for nearly half a millennium, the causative agent (then called *Spirochaeta pallida*) was not isolated by dark field technique by Hoffman and Schaudin until 1905. It was not until the following year that Wasserman developed the complement fixation test for syphilis, based on the earlier work by Bordet and Gengou. Wasserman's work paved the way for more accurate population studies on the incidence of syphi-

lis, and population studies prior to this were based on clinical characteristics of this "great imitator" alone. In light of this, it is difficult to state with accuracy the incidence or prevalence of the disease in the Baltimore population at the time of Poe's death in 1849. Perhaps more importantly, the demonstration of serologic evidence of treponemal infection or the presence of the organism in tissue does not necessarily establish a causal relation with a patient's clinical course or demise, and I would caution against making a generalization regarding mortality statistics based solely on serologic evidence of infection with an organism known to cause indolent and chronic infection.

Parenchymatous neurosyphilis includes general paresis and tabes dorsalis, both of which are chronic in nature and do not generally cause the acute decline in health which has been associated with Mr. Poe's final days. Meningovascular syphilis may cause more dramatic and acute presentation heralded by stroke, although the lack of description of a stroke or more focal neurologic problem in Mr. Poe leads me away from this diagnosis. In addition, there is no description in Poe of hyperreactivity, the Argyll-Robertson pupil, or the dorsal root signs of classic tabes — other typical signs of neurosyphilis. Southard published the case histories of 137 patients with neurosyphilis in 1917, and I would refer those interested in reviewing the more typical findings of parenchymatous and meningovascular syphilis to his manuscript.¹

While delirium, illusion, and hallucinations appear to have been manifestations of the illness that claimed Poe's life, I do not believe that they were the manifestation of neurosyphilis. In addition, I believe Mr. Poe's last years were remarkable for his productivity, including the publication of *The Raven*, *The*

Bells, *Annabel Lee*, *The Casque of Amontillado*, *The Purloined Letter*, *The Masque of the Red Death*, and *The Murders in the Rue Morgue*, all within the last eight years of his life. In my opinion, these were the signs of maturing genius rather than of progressive changes in personality, affect, and intellect as a result of *Treponema pallidum*.

The history of Mr. Poe's chronic consumption of calomel, an inorganic mercuric salt, is fascinating and deserves serious consideration, and we are reminded of Lewis Carroll's "Mad Hatter" as a paradigm for chronic exposure (felt-hat workers were exposed to mercuric salts as well as mercury vapor and often developed erethism). It is unclear to me, however, where the historical references to oliguria, anuria, or "foaming at the mouth" are taken from, and I would be interested in reviewing these. In addition, it should be noted that chronic inorganic mercury exposure usually produces a nephrotic syndrome, rather than the acute tubular necrosis seen in acute poisoning.²

R. MICHAEL BENITEZ, M.D.

Dr. Benitez is assistant professor of medicine, University of Maryland.

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2. Graef JW and Lovejoy FH Jr. Heavy Metal Poisoning, in Harrison's Principles of Internal Medicine, 12th edition, pp. 2185-2186. ■

What Your Patients MAY BE READING

- **Immune to Plague**
Some people can never get AIDS. They are the blessed few, with a genetic inheritance that renders HIV powerless.
Discover, June 1997
- **Gene Therapy: How it will work against cancer, AIDS, Alzheimer's, and more.**
Scientific American, June 1997
- **The AIDS Exception: Privacy vs. Public Health**
It's time to stop granting "civil rights" to HIV — and confront AIDS with more traditional tools of public health
The Atlantic Monthly, June 1997
- **Breast Cancer: the good news**
Potential new weapons in the battle against this baffling disease.
Marie Claire, July 1997
- **Valley of the Dolls Redux?**
Scientists are finally zeroing in on a biological cure for fat. But at what price to your health, heart, and sanity?
Mirabella, May/June 1997
- **Is it a Mole or Cancer?**
How to tell, what to do.
Family Circle, June 24, 1997
- **Running on Empty**
Feeling exhausted, depressed, or mentally fuzzy? You could have an easy to treat thyroid problem. Here's what your doctor may not be telling you.
Health, May/June 1997
- **Stomach trouble?**
You can stop the pain. Pinpoint your symptoms and find a cure that works.
Woman's Day, June 24, 1997
- **Taking Supplements? Easy Does It!**
Unlike other pills, vitamins are sometimes perceived as being as harmless as candy. They're not.
Better Homes and Gardens, June 1997

Speak Out

Ethical Group Capitation

In their January article, Mark S. Seigel, M.D., and Wayne C. Spiggle, M.D., suggested that Med Chi bring together an expert panel to discuss “the issue of capitation as a moral dilemma.” Every payment system in health care has an ethical dimension that physicians should consider carefully, including the many forms of capitation used today.

Some forms of capitation evidently place the best interests of treating physicians and patients in conflict, but others are highly ethical ways to provide quality medical care at an affordable cost – with appropriate compensation for physicians. It would be a mistake to simply oppose all “capitation” – a wide assortment of funding mechanisms – as one entity. Ethical discussions at Med Chi should focus on specific forms of capitation, such as managed care plans that pay individual contracting physicians on a patient-by-patient basis in a manner that substantially affects their individual compensation.

Under ideal conditions, capitation proponents hoped that this funding mechanism would succeed in reversing the economics of health care under the older indemnity/fee-for-service paradigm. Managed care health plans did not emerge in a vacuum. They offered employers – the principal payers in U.S. health care – an alternative to health insurance systems that increased physicians’ incomes at the point patients became sick and received treatment. Under fee-for-service, patients’ large deductibles and coinsurance payments also could deter timely access to primary medical care, and patients had little or no preventive care coverage. After patients satisfied deductibles, physicians had a financial incentive – admitted or not – to use high-cost procedures and hospital-based services that were not medically necessary.

Appropriate and ethical forms of capitation actually create positive financial incentives for physicians to emphasize preventive health care, wellness programs, and timely interventions. They encourage physicians to put in place systems that monitor and assure quality of patient care. They also motivate continuous improvement in patient screening, education, treatment methods, and outcomes.

Consider Kaiser Permanente’s “group capitation” system, in which Kaiser Foundation Health Plans contract with Permanente Medical Groups for a fixed annual amount per patient. The ethical dimension of this system has been explored, debated, and refined by Permanente physicians over many years. Yet, like Drs. Seigel and Spiggle, Permanente physicians see themselves as uncompromising advocates of “what is right for patients.”

Speak Out

Because Permanente physicians receive compensation primarily through salaries paid by our group, they practice medicine without worry that individual patient care decisions will affect their personal income. They also share responsibility for prudent, ethical, and medically appropriate use of the medical group's resources. Unlike systems that could reward undertreatment financially, Permanente physicians are eligible to receive a very small additional incentive for meeting the group's annual quality, service, and cost-effectiveness goals. Neither salaries nor incentives are linked to individual patient care decisions.

Our group capitation structure – reviewed periodically to encourage positive physician behaviors defined by their peers – properly aligns the quality of care, attention to preventive services, clinical outcomes, and financial goals. The Permanente medical group – not the individual physician – receives payment for comprehensive care in advance.

Group capitation has been a fundamental tenet of our integrated health care delivery system. It supports excellent relationships among our primary care physicians, our specialists, and community specialists. And it builds on the premise that high quality care – such as timely referrals to specialists and continuity of care – ultimately produces lower costs and eliminates the need for more costly care later.

ADRIAN E. LONG, M.D.

Dr. Long is the president and medical director of Mid-Atlantic Permanente Medical Group, P.C. based in Rockville, Maryland. ■

EDITORIAL COMMENT: *This speakout is a reminder for all of us that no matter what the "system," medical care as we know it requires individual ethical decisions. While the author espouses the Permanente Medical Group, it is wise to remember that you are working for the name on your paycheck – whether it be Medicare, HMO, commercial insurer, or the patient. Any physician-patient relationship is complex and offers the risk of over- or undertreatment. However, that risk increases with the number of intermediaries. There may not be an ideal arrangement, but we must continue the dialogue because, sooner or later, each of us will need the advice and care of a physician.*

JOHN BUCKLEY, M.D., member, editorial board ■

Ross J. Van Antwerp, D.O., M.P.H., and **Francis M. Giardello, M.D.,** are among the authors of "Colorectal Cancer Screening: Clinical Guidelines and Rationale," published in *Gastroenterology* (1997;112:594-642). The article provides recommendations regarding colorectal cancer screening and surveillance for those at average risk and those at increased risk. The recommendations are intended to guide physicians, and the public, in decision making. Dr. Van Antwerp is in private practice at the Laser Center of Maryland, Severna Park, Maryland. Dr. Giardello is director, Hereditary Colon Cancer and Polyposis Clinic at the Johns Hopkins University.

Lawrence Appel, M.D., is lead author of a study published in the April 17 issue of *The New England Journal of Medicine* that once again reports that a diet with more fruits and vegetables will substantially and quickly lower blood pressure. The study included 459 adults who were given one of three diets for eight weeks. Dr. Appel is an associate professor of medicine at the Johns Hopkins University.

Baltimore psychiatrist **Frank J. Ayd, Jr., M.D.,** is the editor and author of one of thirteen chapters of *The Art of Rational Risperidone Pharmacotherapy*. This monograph, published by Ayd Medical Communications, is based on the proceedings of symposiums held in Baltimore in October 1995 and January 1996, in which renowned experts on risperidone spoke on the use of risperidone in children and adolescents, and its use in treatment-refractory obsessive compulsive disorder and autism spectrum illnesses. Dr. Ayd is emeritus director of professional education at Taylor Manor Hospital in Ellicott City, Maryland.

Rudolf Trawoger, M.D., Theodor Kolobow, M.D., Maurizio Cereda, M.D., and Maria Elena Sparacino, M.D., are coauthors of "Tracheal Mucus Velocity Remains Normal in Healthy Sheep Intubated with a New Endotracheal Tube with a Novel Laryngeal Seal," recently published in *Anesthesiology* (1997;86:1140-1144). Dr. Kolobow is chief, and Drs. Trawoger, Cereda, and Sparacino are visiting fellows, section of pulmonary and cardiac assist devices, pulmonary/critical care medicine branch, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland.

James C. King, M.D., was the principal investigator in a multi-center trial that showed a new flu vaccine given to children via nose drops or a nasal spray was safe and produced good antibody response. The study included 356 children between the ages of 18 months and six years and lasted from the fall of 1995 to the spring of 1996. Results were recently published in the April issue of *Pediatric Research Journal*. Dr. King is associate professor of pediatrics at the University of Maryland Medical Center.

H. Ballentine Carter, M.D., is lead author in a study, published in *The Journal of the American Medical Association* (May 13, 1997), that shows men with no suspicious signs of prostate cancer on rectal examinations, who have prostate specific antigen (PSA) levels below 2.0, could safely wait two years before their next PSA test. Dr. Carter and colleagues examined PSA measurements of 312 men in the Baltimore Longitudinal Study of Aging. Dr. Carter is from The Johns Hopkins University School of Medicine, Baltimore.

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Preventing wading pool suction-drain injuries

Elizabeth S. Porter, M.D., M.P.H., Ingrid C. Kohlstadt, M.D., M.P.H.
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Drs. Porter and Kohlstadt are preventive medicine residents at The Johns Hopkins University School of Hygiene and Public Health.

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ABSTRACT: *Wading pools with displaced drain covers and single drain outlets can lead to life-threatening injuries. This is a case of a Maryland child who sustained transanal suction which resulted in prolapse and avulsion of the small intestine from its blood supply. Because the injury would likely have been prevented if at least one of three safety precautions had been followed, standards for pool operation and pool design are reviewed.*

W, a healthy three-year-old boy, was playing in a public wading pool. He sat on an uncovered central drain, and his perineum formed a vacuum seal with the drain. Supervising adults noticed W's discomfort but they were unable to lift him off the drain until the valve to the wading pool pump was closed. Upon rescue, it was apparent that he had sustained serious internal injury, necessitating emergency transport to a tertiary care hospital. His rectum was prolapsed, and the small intestine was avulsed from its blood supply. Extensive surgical resection of his small bowel was required. W will have a life-time disability from "short-gut syndrome." He may be a future candidate for small bowel transplantation.

Though uncommon, suction-drain injury has been described in the literature, and can be prevented.^{1,3,4} The Anne Arundel County Department of Health in Maryland summarizes three missed prevention opportunities surrounding the case of W, and reviews pool safety measures aimed at primary prevention of similar suction-drain injuries in children.

Wading pools allow toddlers to play independently under adult supervision. Wading pools can be filled with water to a depth no greater than 18 inches, shallow enough for children to play seated. W was sitting in the center of the pool, on top of the single functioning drain. Drains require

grated covers to prevent suction seal; however, the drain on which W sat was uncovered. The suction force caused relaxation of the anal sphincter and subsequent internal damage. To prevent suction-drain injury, The American National Standards Institute/National Spa and Pool Institute² specify that all drain outlets be covered with undamaged grates, secured so they can only be removed with tools. Any type of drain cover is somewhat effective, particularly when the size is greater than 12" x 12", the perforations cover a large area, and the surface is convex upwards making blockage unlikely. The use of semi-permanent anti-vortex drain covers has been recommended, especially for pools with a single drain or multiple drains that can be isolated by valves. However, experience with these injuries in North Carolina has shown that anti-vortex drain covers are ineffective by themselves because drain covers are too easily damaged or removed.⁴ Pool operators or maintenance personnel should inspect pool drains daily to assure that covers are properly secured and in good condition. Wading pools should be closed if drain covers are missing, broken, or inadequately secured.⁴

The second missed prevention opportunity involved the pumping system. To maintain cleanliness and disinfection in wading pools, water must recirculate every two hours. The pool pump is connected to two outlets: a central drain and a surface skimmer. For pools constructed or remodeled since 1992 in Maryland, these two outlets must be interconnected such that if one outlet is occluded, the pump can still draw from the other. Older pools may have only one outlet or outlets that can be easily isolated, making suction-drain injuries a greater risk. The pool that W used was in disrepair with only one suction-outlet in operation. Pool operators had previously noted the surface skimmer had a significant leak, and a decision was made to disconnect it until repairs could be made after the season. As such, the central drain was the only suction outlet operating. The engineering aspects of pool drainage systems deserve careful attention by pool operators and licensing agencies. Pumping systems in new pools are designed to prevent suction-drain injuries. A pressure-limiting switch has been designed to cut power to the pump if a vacuum occurs. A switch to shut off the pump for a displaced drain cover is also under consideration.

In the case of W, a third missed prevention opportunity was the pool attendant was not a certified lifeguard. Pool

operators, lifeguards, and other pool personnel must know how to shut off the pump and must have ready access to pump switches. W's injury would have been mitigated by quicker response; however, the importance of primary prevention rather than early detection should be emphasized. Subsequent inquiries by the health department suggested lifeguards, pool operators, and inspectors are generally familiar with the regulations, but not with the rationale behind them, increasing the potential for suction-entrapment injuries. Case information is now emphasized in training courses for pool operators and pool inspectors in Anne Arundel County. In addition, companies which install and service pools in this county will receive a fact sheet to encourage careful attention to preventive engineering practices.

W's suction-drain injury does not stand in isolation. Drownings from hair entrapment and suction injuries from pool cleaners are also missed opportunities for prevention. We emphasize the need for engineering controls, vigilant pool maintenance, and employee training through case examples.

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Dengue fever: A risk to travelers

Beth E. Karp, D.V.M., M.P.H.

Dr. Karp is an epidemiologist in the division of outbreak investigation in the Epidemiology and Disease Control Program at the Maryland Department of Health and Mental Hygiene.

ABSTRACT: *An outbreak of dengue fever occurred among a small group of Maryland and Pennsylvania residents following a trip to the British Virgin Islands in January 1996. Dengue fever is a mosquito-borne viral illness that occurs primarily in tropical urban areas. Most dengue infections are benign and self-limited, but some produce severe and fatal hemorrhagic disease. Although dengue is not endemic in the continental United States, travelers may acquire the infection during visits to the tropics. Physicians should consider dengue in the differential diagnosis of a patient with a febrile illness and a history of recent travel to a tropical area. Travelers to endemic areas should be advised to take precautions to prevent mosquito bites.*

Dengue fever, also known as breakbone fever, is an acute mosquito-borne febrile illness caused by four dengue virus serotypes. Dengue affects more people worldwide than any other arthropod-borne disease except malaria,¹ and it is regarded as the most important arboviral disease of humans.²⁻⁴ Although not endemic in the 50 United States, dengue is a risk to residents who travel to endemic areas.⁵ This report describes an outbreak of dengue fever in a group of travelers returning from the Virgin Islands. Clinical and epidemiologic aspects of dengue are reviewed and prevention strategies are briefly discussed.

An imported outbreak: January 1996 to February 1996

In January 1996, a group of eight adults, including six Maryland and two Pennsylvania residents, traveled to the island of Jost Van Dyke in the British Virgin Islands for a ten-day vacation. The group consisted of five men and three women ranging in age from 27 years to 52 years (median

38 years). The group arrived in Jost Van Dyke on January 19 and departed on January 28, traveling via the airport in St. Thomas. The group's main activities were swimming and snorkeling. In addition, five party members took a day trip to the island of Tortola. Party members stayed at a Jost Van Dyke camp site, where they slept in four screened huts without air conditioning or bed nets, drank bottled water, and used rainwater for cooking. One party member noted the presence of an open rain water bucket that had a film of "slime" on top. The travelers applied topically a commercial bath oil to repel mosquitoes. In addition, one couple sprayed an N,N-diethyl-m-toluamide (DEET) containing insect repellent around the windows of their hut. Several members of the group reported they were bitten by mosquitoes.

During the week following the trip, six group members (three men and three women) developed a febrile illness. Three developed symptoms on January 28, while the other three became symptomatic on February 1, 2, and 3. Signs and symptoms included fever (six), chills (six), myalgias (six), eye pain (five), headaches (four), arthralgias (four), nausea or vomiting (four), cough (three), sore throat (three), rash (two), diarrhea (two), nasal congestion (one), and epistaxis (one). Four cases reported a fever of 104°F or higher. Four of the ill sought medical attention, but none were hospitalized. The duration of illness ranged from four days to approximately two weeks, with some party members reporting residual symptoms for several more weeks.

The Pennsylvania and Maryland departments of health became aware of the outbreak in late February and arranged for interviews of all party members. An infectious disease specialist consulted by one of the travelers had suspected dengue fever and submitted serum samples from two of the ill to the Centers for Disease Control and Prevention (CDC). Subsequently, the other six party members submitted blood samples, through their local health departments, to the CDC for dengue testing. Serum specimens from all of the travelers were tested at the CDC Dengue Branch in Puerto Rico, using an enzyme-linked immunosorbent assay (ELISA), for anti-dengue IgM antibody to a mixture of four dengue virus antigens. Seven of the serum samples were positive for anti-dengue IgM antibodies and one was negative. Consequently, there were six symptomatic dengue cases and one asymptomatic dengue infection among the eight travelers. Although the ELISA test does not distinguish among dengue serotypes, it is known from other samples that dengue type one was active in the British Virgin Islands at the time of this outbreak.

Clinical aspects of dengue

The spectrum of illness associated with dengue virus infection is broad, ranging from subclinical infection or mild disease to severe disease and death. Symptoms begin 3 to 15

days after the bite of an infective mosquito.³ Classic dengue fever is characterized by a sudden onset of high fever, severe headache, myalgias, and arthralgias.² The musculoskeletal pains associated with dengue infection are often severe, hence the name "breakbone fever."⁶ Retrobulbar pain accentuated by eye movement is a distinctive feature of dengue. Other common signs and symptoms include nausea, vomiting, rash, and conjunctivitis. Lymphadenopathy, hepatomegaly, and mild hemorrhagic manifestations such as epistaxis, may occur. Other, less common features have been described. Dengue fever is commonly confused with a variety of febrile illnesses including influenza and measles.^{3,7}

Dengue hemorrhagic fever (DHF) is a severe form of dengue that is characterized by abnormal hemostasis and vascular permeability.³ Based on the World Health Organization case definition, a case of DHF must have fever, minor or major hemorrhagic manifestations, thrombocytopenia (platelet count $\leq 100,000/\text{mm}^3$), and objective evidence of increased capillary permeability (hemoconcentration, pleural effusions, or hypoproteinemia). Dengue shock syndrome (DSS) occurs when a case meets all of the criteria for DHF and has hypotension or narrow pulse pressure.^{5,8}

Health care providers should consider dengue in the differential diagnosis of a patient with a febrile illness and a history of recent travel to a tropical area. The diagnosis can be confirmed by isolating the virus from a serum sample obtained within the first five days of illness or by demonstrating a four-fold rise in dengue-specific antibodies in paired serum samples (acute and convalescent). A presumptive laboratory diagnosis can also be made by detecting anti-dengue IgM antibody by an ELISA test. Suspected dengue cases should be reported to the appropriate state or territorial health department. Serum specimens may be sent for confirmation to the CDC's Dengue Branch through the state or territorial health department laboratory. Specimens should be accompanied by a clinical summary and a detailed travel history. The dates of illness onset and blood collection should also be included.^{3,5,7}

There are no specific treatments for dengue infections and no vaccines are available. Classic dengue is usually benign and self-limited, with fever typically lasting three to seven days. Convalescence may be prolonged.^{1,2} Treatment for classic dengue is supportive. The patient should be encouraged to rest and drink plenty of fluids. Aspirin should be avoided due to its anticoagulant effects and the increased risk of Reye's Syndrome.^{3,7} The patient's blood pressure, hematocrit, and platelet count should be monitored.⁵ Aggressive intravenous fluid replacement is essential in the treatment of patients with DHF/DSS.^{3,6,7} The fatality rate for patients with DSS can be as high as 44%, while appropriately treated DHF cases have a fatality rate of 1% to 2%.⁹

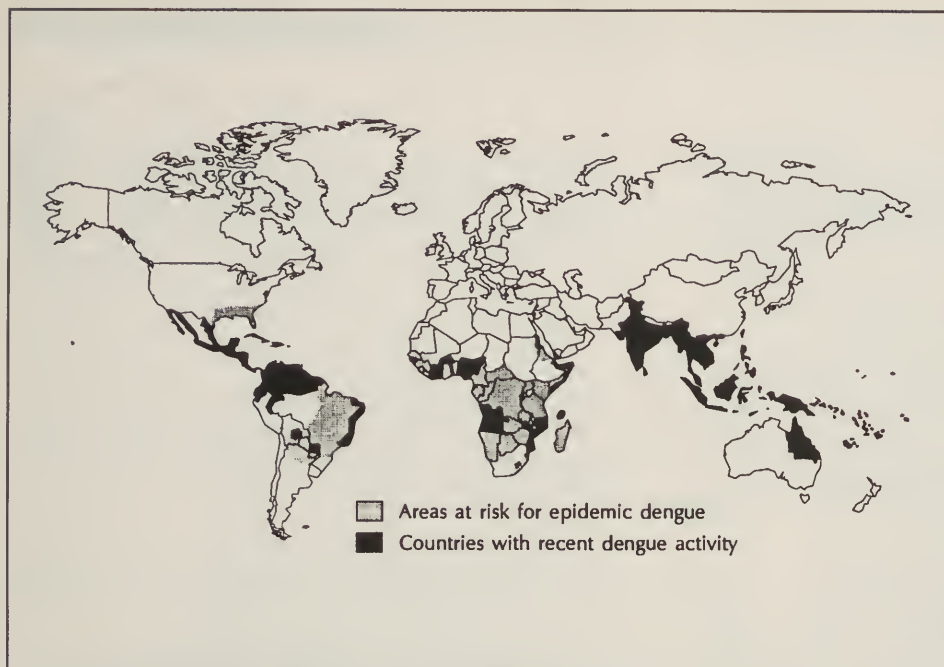


Figure 1. Worldwide distribution of dengue viruses and their primary mosquito vector, *Aedes aegypti*, 1995.

From: CDC. Health Information for International Travel, 1995.
Atlanta: HHS publication no.(CDC) 95-8280, August 1995.

Epidemiology of dengue

Dengue fever is caused by four closely related, but antigenically distinct, virus serotypes of the genus *Flavivirus*.¹⁰ Virus transmission occurs through the bite of infective female *Aedes* mosquitoes. A female mosquito becomes infected by biting an infected human during the viremic period, which lasts for about five days. The mosquito can transmit the virus to humans after an extrinsic incubation period in the mosquito of 8 to 12 days.^{7,10} Dengue, unlike many other arboviruses, is not zoonotic in urban areas, and with few exceptions, has no avian or nonhuman mammalian reservoirs.⁶ The primary vector mosquito, *Aedes aegypti*, is also the principal urban vector of yellow fever.⁷ It is the only documented vector of dengue in the Americas.¹¹ *Aedes aegypti* mosquitoes, which are found most frequently in or near human habitations, prefer to feed on humans during the daytime, with peak biting activity in the early morning and late afternoon. They breed primarily in man-made water holding containers such as discarded tires, barrels, buckets, flower pots, and cans.² *Aedes albopictus*, the Asian tiger mosquito, is responsible for endemic transmission of dengue in Asia.⁴

Dengue is primarily an urban disease of the tropics.¹⁰ Dengue viruses are endemic in most tropical countries of the South Pacific, Asia, Caribbean, Mexico, South and Central America, and Africa (**Figure 1**). Dengue is rapidly expanding in most tropical areas of the world, with millions of cases occurring each year.² The emergence of dengue as a significant public health problem has been facilitated by demo-

graphic changes such as increased urbanization and rapid population growth, poor sanitation, increased air travel, and ineffective mosquito control programs.^{1,10}

DHF, which was first described in the 1950s,³ has increased dramatically in incidence over the past 20 years. Although not well understood, data suggest that age, virus strain, immune status, and genetic background are important risk factors for DHF/DSS. DHF is most common in children. International travelers from nonendemic areas such as the continental United States, are thought to be at low risk for severe infection.² Recovery from infection with one serotype provides long-term homologous immunity, but does not provide durable cross-protective immunity against other dengue virus serotypes. In fact, the risk of de-

veloping the more severe form of the disease is significantly higher after a second dengue infection. This phenomenon, which has significant implications for vaccine development, has been explained by a theory known as immune enhancement. In this proposed mechanism, nonneutralizing antibodies form complexes with the virus, thereby enhancing entry of the virus into mononuclear phagocytes. This results in increased activation of complement and kinins and the release of mediators of vascular permeability.^{4,7}

Although dengue is primarily a disease of the tropics, U.S. health care providers should be concerned about dengue for several reasons. First, dengue infections may be encountered among travelers arriving from endemic areas.⁷ The risk of infection among travelers appears to be small, unless an epidemic is in progress. However, imported cases are reported each year.² From 1977 to 1994, 2248 suspected cases of imported dengue were reported in the U.S. Approximately 481 of these cases were confirmed as dengue.¹⁰ In 1995, 448 suspected cases of imported dengue were reported in the U.S. and 86 of these cases were laboratory-diagnosed. Based upon travel histories available for 81 of the laboratory-diagnosed 1995 cases, infections were probably acquired in the Caribbean islands (48), Mexico and Central America (24), Asia (five), South America (three), and Africa (one).⁹ It is likely that many more cases go undetected each year because dengue surveillance in the U.S. is passive, dependent upon people to seek medical attention and physicians to recognize and report the disease.^{5,10}

A second reason for concern is the small, but significant, risk of dengue transmission in the United States. Currently, two competent mosquito vectors, *Ae. aegypti* and *Ae. albopictus*, are present in the U.S.^{7,10} In Maryland, *Ae. aegypti* has been found sporadically in low numbers, while *Ae. albopictus* infestations have been detected in several counties.¹² Historically, one of the earliest dengue epidemics described in the medical literature occurred in Philadelphia in 1780. The southeastern U.S. was plagued by dengue epidemics through the nineteenth and twentieth centuries, most recently in Louisiana in 1945.⁷ Indigenous transmission also occurred in south Texas in 1980, 1986, and 1995 as a result of outbreaks in northern Mexico.^{7,9,10,13} Dengue viruses have also been active in U.S. territories and commonwealths, including the U.S. Virgin Islands and Puerto Rico.⁵

Prevention and control of dengue

Ae. aegypti, the principal dengue vector, lives in close association with its human hosts, breeding primarily in man-made water holding containers.⁷ In recent years, public health authority recommendations for dengue prevention have emphasized mosquito control through sustainable, community-based programs to reduce mosquito breeding sites, with limited reliance on chemical larvicides and adulticides.^{5,7,10} It is likely that this approach will take some time to significantly impact disease transmission.¹⁰ Currently, the only effective way to avoid dengue infection in endemic areas is to avoid mosquito bites. Travelers can reduce their risk of infection by remaining in air conditioned or well-screened areas when possible, by wearing clothing that adequately covers the extremities, and by applying mosquito repellent. The most effective repellents are those containing 20% to 30% DEET. Higher concentrations of DEET may cause side effects, particularly in children. Permethrin or DEET repellents can be sprayed onto clothing to prevent mosquitoes from biting through thin garments.^{2,14} If bedrooms are not air conditioned or well-screened, the use of insecticidal sprays and bed nets is recommended.^{3,14} An article published in 1993 addressed the safety of DEET and rated the effectiveness of 21 commercial products that are used to repel insects.¹⁵

Currently, there are no approved dengue vaccines available. Research continues on developing a tetravalent vaccine that is both safe and effective.⁷ Novel vector control

strategies, such as the genetic alteration of mosquitoes, are also under investigation.¹⁶

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A BROCHURE ENTITLED "Preventing Dengue Fever in Travelers," may be obtained from the Centers for Disease Control and Prevention, Dengue Branch, 2 Calle Casia, San Juan, Puerto Rico 00921-3200. Single copies of the brochure are also available from the Maryland Department of Health and Mental Hygiene, Epidemiology and Disease Control Program, 201 West Preston Street, Baltimore, Maryland 21201, (410) 767-6677.

Geriatrics for the Clinician

OSTEOPOROSIS: A FOCUS ON TREATMENT

Introduction

Osteoporosis is characterized by decreased bone mineral density (BMD). The principle result of this decreased density is a greatly increased risk for fractures.¹ Osteoporosis affects approximately 25 million individuals in the United States, mostly women.² In fact, one in four women over the age of 60 years has osteoporosis. Men are also affected by osteoporosis, but due to higher average BMD and decreased life expectancies, their risk for osteoporosis-related fractures is much lower. One of the goals in managing patients with osteoporosis is to decrease the risk of fractures, especially hip fractures. Although less common than vertebral fractures, hip fractures have been associated with greater loss of physical function, cost (lengthy hospital stays, nursing home admissions), and mortality. Nineteen percent of patients with a hip fracture (1.9% with a vertebral fracture) will require long-term care in a nursing facility.³ In one

year, the cost of medical care and rehabilitation for osteoporosis-related fractures was estimated at 5.1 billion dollars.⁴

With our population increasing in age, the impact of this disease on the individual and society will be tremendous. This article focuses on the treatment of this potentially devastating problem in postmenopausal women.

Osteoporosis risk assessment

Until age 40, bone formation equals bone resorption, but beyond 40 years of age, resorption exceeds formation by approximately 0.5% per year — age-related bone loss.¹ In women, 1% to 1.5% of total bone mass is lost each year after the last menstrual period (LMP) — postmenopausal-related bone loss.¹ Thus, postmenopausal women are already at significant risk for osteoporosis based on a combination of their age and hormone status. Many other factors impact bone mass (Table 1). Together these factors can increase a person's chance of developing osteoporosis, but they are not predictive of the development of the disease.

Diagnosis

Pain, spinal deformity, and height loss are common signs and symptoms of osteoporosis. The diagnosis is based on the clinical picture or radiographic tests. The dual energy x-ray absorptiometry (DEXA) is considered to be the gold standard for diagnosing osteoporosis⁵ (Table 2). Prophylaxis may be indicated to prevent future fractures with osteopenia, whereas intervention is indicated once a patient is diagnosed with osteoporosis.⁶

Treatment/approaches

After determining a patient has osteoporosis, several options exist. Non-pharmacologic approaches include changing modifiable risk fac-

Table 1. Risk factors for osteoporosis

Non-modifiable risk factors	Modifiable risk factors
<ul style="list-style-type: none"> • Low peak BMD • Increased age • Female gender • Asian, Caucasian race • Family history • Small frame • Early menopause 	<ul style="list-style-type: none"> • Cigarette smoking • Inadequate calcium intake • Sedentary lifestyle • Medications: <ul style="list-style-type: none"> • aluminum antacids • carbamazepine • GnRH agonists* • levothyroxine • Substance use: <ul style="list-style-type: none"> • caffeine • Disease states: <ul style="list-style-type: none"> • Depression • Primary hyperparathyroidism • glucocorticoids • heparin • phenytoin • ethanol • Hyperthyroidism

*GnRH = gonadatropin releasing hormone

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tors (Table 1) and preventing falls. All patients should receive counseling about their modifiable risk factors. Weight bearing exercise, such as walking, stair climbing, dancing, or tennis, should be initiated.⁷ Patients should quit smoking and avoid excessive alcohol and caffeine consumption. Also, clinicians should take caution when prescribing thyroid hormone or corticosteroids as these medications can increase bone loss.

Preventing falls is another important non-pharmacologic approach. Patients with hearing or vision impairment, vestibular dysfunction, and cognitive decline are at an increased risk for falls. Medications associated with falls include benzodiazepines, barbiturates, diuretics, and antidepressants. The patient's environment should be made as safe as possible, for example, proper lighting, floor coverings (non-skid rugs), and handrails.

Along with non-pharmacologic approaches, patients with osteoporosis may benefit from pharmacologic therapy. All post-menopausal women should receive 1000mg to 1500mg of calcium daily.⁸ If patients are not receiving enough through their diet, supplemental calcium can be taken to achieve the recommended daily calcium intake. Along with calcium, vitamin D is essential in the maintenance of healthy bone. Patients should receive 400 IU of dietary vitamin D daily. Patients should not receive higher doses or therapy with more active metabolites of vitamin D unless a specific disorder of vitamin D metabolism exists, such as renal or liver disease.

In addition to receiving adequate calcium and vitamin D, adjunctive therapies such as

estrogen, alendronate, salmon-calcitonin, and slow-release fluoride exist. All of these agents have been shown to increase BMD, and more specifically, BMD at the hip, a surrogate marker which has been shown to correlate well with fractures.⁹

Estrogen

Estrogen should be considered the agent of choice in the treatment of postmenopausal women with osteoporosis. Estrogen increases BMD (5% to 10% vertebral, 5% hip) and decreases the incidence of vertebral and hip fractures by about 50%.¹⁰⁻¹² Estrogen also decreases the symptoms of menopause and protects against coronary heart disease. Although the benefits are greatest when estrogen is taken during or immediately after menopause, women averaging 14 years past menopause have benefited. The minimally effective dose for estrogen is 0.625mg daily of conjugated estrogens.¹³ Adverse, but minor, effects of estrogen therapy include breast tenderness, edema, nausea, and vomiting. Women with an intact uterus will require combination hormone therapy (estrogen and progestin) to decrease the risk of endometrial cancer. Single tablet formulations of estrogen and progestin are available for convenience. Progestins have minimal to no effect on BMD.

Despite all of the advantages, not all patients are candidates for estrogen therapy. There is still some concern regarding an increased risk of breast

cancer in estrogen users.¹⁴ Other concerns with estrogen therapy are less controversial. Patients with hepatic insufficiency or thromboembolic disorders should avoid taking estrogen.

Table 2. Densitometric criteria for bone mass status

Bone Mass Status	Relative BMD ^a
Normal	<1 SD ^b below mean ^c
Osteopenia	1-2.5 SD ^b below mean ^c
Osteoporosis	>2.5 SD ^b below mean ^c

^a BMD = bone mineral density

^b SD = standard deviation

^c Mean value reference value in pre-menopausal Caucasian women

For patients whom estrogen is contraindicated, second-line agents include alendronate and salmon-calcitonin.

Alendronate

Alendronate, the offspring of etidronate, the original bisphosphonate, has been associated with

significant increases in BMD: 7% to 9% at the spine and 6% at the hip with 10mg of alendronate daily.^{15,16} Unlike its parent compound, etidronate, alendronate demonstrated normal bone mineralization.¹⁵ Alendronate has also been found to decrease the number of vertebral and hip fractures in post-menopausal women with osteoporosis receiving supplemental calcium.¹⁵⁻¹⁷ In one of these studies, alendronate decreased the risk of hip fracture by 50% compared to placebo.¹⁷

The concern with alendronate has been its adverse effect profile. Several case reports of erosive esophagitis have been reported.^{19,20} Approximately 25% of these were serious enough to require hospitalization or temporarily disable the patient. These adverse effects typically occurred within two months of alendronate initiation and resolved with cessation of therapy and a combination of acid suppression therapy, analgesia, and/or parenteral nutrition. The long-term complication associated with these adverse events is unknown, and patients should be followed to assess for the development of strictures. Three issues were raised: 1) non-adherence with administration guidelines, 2) continuation of therapy after onset of symptoms, and 3) pre-existence of esophageal disorders.¹⁹ To minimize the risk of esophageal erosion, alendronate must be taken after the pa-

Table 3. Administration guidelines for alendronate

1. Take when awake, moving about, and upright each morning.
2. Do NOT take at bedtime or before rising.
3. Take tablet with 8 ounces of plain tap water at least 30 minutes before the first food of the day.
4. Do NOT take medications, food, or beverages with your alendronate tablet. (This may decrease absorption.)
5. Do NOT lie down for at least 30 minutes after swallowing the tablet and after the first meal or food of the day.
6. If you forget to take your tablet in the morning, do NOT take the tablet later that day — skip that day.

tient is awake, moving about, and in an upright position (Table 3). Because it may be difficult for bedridden patients to comply with these administration guidelines, alendronate should not be used in these patients. Other common complaints include nausea, dyspepsia, gastritis, abdominal discomfort, and pain. Due to alendronate's adverse gastrointestinal (GI) effects, it should be avoided in patients who have a history of dysphagia or other GI-related diseases. Additionally, women with low levels of calcium or renal insufficiency (Clcr <35mL/min) are also advised not to take alendronate.

With only three years of published data, the long-term effects of alendronate on bone turnover and quality of bone are still unclear.

Calcitonin

Salmon-calcitonin administered in doses of 200 IU/day intranasally, five to seven times a week, demonstrated an increase of 2% to 3% in BMD at vertebral sites.^{21,22} In addition to increasing vertebral BMD, salmon-calcitonin given intranasally 200 IU daily has also been found to decrease vertebral fractures.²³ Unlike the earlier injectable product, intranasal salmon-calcitonin offers the advantages of fewer adverse effects ($\geq 10\%$ nausea and vomiting with intramuscular SCT), less expensive, and non-injectable (patient acceptance, ease, adherence). Salmon-calcitonin may have greater benefit for

patients who are more than five years past menopause. Subgroup analysis in one study demonstrated greater benefit in late postmenopausal women (≥ 5 years since LMP) compared to women within five years of menopause with intranasal salmon-calcitonin 200 IU daily.²²

Sodium fluoride

Sodium fluoride is the only agent which acts to increase bone formation. All of the other agents discussed suppress bone resorption. Slow-release sodium fluoride has been recently shown to increase BMD at the spine (4% to 5%) and hip (2% to 3%).²⁴ There is some evidence that BMD increases in the 15% to 20% range (over four years of treatment) are possible. However, one in four patients do not respond and fluoride's effects are mainly on vertebral (trabecular), not hip bone (cortical). At this time, there is not enough evidence to recommend sodium fluoride; outcome studies examining the effect of this agent on hip fracture must be performed.

Conclusion

All of these agents have been shown to increase BMD and decrease the incidence of fractures. Estrogen should be considered first line therapy. Estrogen provides protection against cardiovascular disease, alleviates the symptoms of menopause, and is less expensive than the other agents. For patients with contraindications to estrogen therapy, or who choose not to take estrogen, alendronate and salmon-calcitonin are alternatives. Slow-release sodium fluoride is a promising therapeutic agent, but to date, insufficient evidence prevents its recommendation. Therapy with these agents is considered life-long and patients should be monitored for effectiveness (fractures, height loss, spinal deformity, DEXA) and toxicities of the specific agents.

Osteoporosis can be a debilitating disease with significant and costly consequences. If individuals who are at greatest risk are screened for low BMD, the incidence of fractures may be decreased by placing these women on effective anti-fracture therapy.

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A rational approach to psychological testing of adults with mental retardation

by Jerome H. Feldstein, Ph.D.

Abstract

Intelligence (IQ) tests and scales of adaptive behavior are typically used to evaluate adults with mental retardation. Personality tests and instruments designed to measure behavior problems and psychopathology are also used. Repeated IQ testing is common but not useful for adults. Adaptive behavior scales and measures of psychopathology do appear useful, although the latter are relatively new and not widely used in clinical practice. Tests requiring skilled language responses are not useful for people with severe and profound disabilities. The problem of administering the tests is addressed by interviewing people who are knowledgeable about the person being evaluated; this method is limited by the actual knowledge of the person interviewed. Neuroimaging, still in the research stage, may be especially relevant in the future.

People with mental retardation are subjected to more psychological testing than most other Americans; IQ testing is the most frequent type. An IQ test is required to establish a diagnosis of mental retardation. However, it is common practice to repeat testing on a regular basis, possibly over many years. While this may be useful in childhood, especially for school placement decisions, much of the testing performed on adults serves no purpose. In contrast, scales of adaptive behavior and tests of psychopathology may be underutilized in adults with mental retardation. This article is organized by common test types and discusses the usefulness of each type of test when evaluating adults who have mental retardation. Commonly used tests are mentioned, but an extensive listing of tests is not attempted. Any such list would soon be out of date.

IQ Tests

Tests of "intelligence" are integral to the diagnosis of mental retardation. These tests were originally developed to identify children with mental retardation, then called "feeble-minded," prior to their failing in school.¹ However, the relationship of such tests to the construct of "intelligence" is controversial. The goal of an IQ test is to obtain a score that serves as the major factor in mental retardation diagnosis. The most important of these tests are the Wechsler Adult Intelligence Scale, Revised (WAIS-R)² and the Stanford-Binet, Four.³ Another widely used test is the Slosson Intelligence Test (SIT),⁴ which is popular because of the relatively brief time needed for administration. The validity of other tests

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This article is the second in a series that will explore various aspects of treating patients with developmental disabilities.

on the market is described in terms of their correlation with either the Wechsler or Stanford-Binet. That is, test makers claim their products are true measures of intelligence if what the tests measure is substantially similar to what is measured by the Wechsler or Stanford-Binet tests.

The most recent definition of mental retardation is:

Mental retardation refers to substantial limitations in present functioning. It is characterized by significantly subaverage intellectual functioning, existing concurrently with related limitations in two or more of the following applicable adaptive skill areas: communication, self-care, home living, social skills, community use, self-direction, health and safety, functional academics, leisure, and work. Mental retardation manifests before age 18.^{5,6}

The definition contains three elements, as did its predecessor, the first of which refers to intellectual functioning, defined as "an IQ standard score of approximately 70 to 75 or below, based on assessment that includes one or more individually administered general intelligence tests developed for the purpose of assessing intellectual functioning."^{5,6} Thus, an IQ test is necessary, but not sufficient, for making a diagnosis of mental retardation. However, the diagnosis must be made before the age of 18 years. Adults with mental retardation have already been diagnosed.

Are there any reasons to give IQ tests to adults with mental retardation? One possible reason is to investigate a suspected decline in cognitive functioning. Such a decline may result from illness, accident, or dementia. Since IQ tests are constructed to yield stable scores over time, a significant performance decline could indicate a serious health problem. However, this would only apply to people with mild or moderate retardation. People with severe or profound disabilities have scores too close to the test floor to show large decrements; many of them cannot be tested at all. Test scores can also be affected by motivational variables. A social psychology study of people with mental retardation, in an institution, demonstrated motivational effects.⁷ In that study, patients were able to raise their scores when told a higher score would enable entry into a desirable new program. When told the new program was not desirable, scores declined.

A less likely scenario could involve the reversal of a diagnosis of mental retardation. While a diagnosis may be reversed, it is not likely to occur as the result of IQ testing. A person without limitations in adaptive skills, despite a relatively low IQ score, may drop out of the mental retardation service system, particularly after leaving school. IQ

tests are basically academic in nature; therefore, a person without need for many academic skills in daily life would not be impaired after leaving school.

The most common reason for IQ testing of adults is bureaucratic requirement. People living in institutions may be tested regularly, possibly every year. In Maryland, people leaving institutions must have a recent IQ test to be accepted into a community residence. There is no apparent reason for this requirement; test scores are not used to make placement decisions. School systems require such information for academic placements, but there is no parallel with adults.

People with the most severe disabilities, traditionally characterized as profoundly retarded and multiply-handicapped, do not have the skills to participate in standard IQ tests. Sometimes, such people are evaluated with scales of infant development. The items on these scales involve sensorimotor skills because they are written for pre-verbal children. This type of testing serves no purpose, other than producing a score. It is of no use to compare motor performance of physically impaired adults to that of typical infants. Comparisons are only meaningful if they are made between persons at a similar point in life. The fact that an adult performs certain motor tasks in a manner similar to a young infant is completely irrelevant to the assessment of general intellectual functioning.

Does IQ testing do any harm? It might. First of all, the testing situation is not necessarily a pleasant experience. Persons tend to find situations in which they experience frequent failure to be aversive; that is the case when people with mental retardation are given a series of academic tasks. IQ test instructions require only neutral feedback be given, even when the patient asks about the correctness of an answer. Thus, people who are not good at academics are subjected to a long, possibly tiring, series of academic tasks without positive feedback. A more insidious problem involves the interpretation of IQ scores by professionals and paraprofessionals who are responsible for provision of services. People with mental retardation may be denied training opportunities when gatekeepers determine particular IQ scores preclude their profiting from such training. The only way to determine a person will not profit from training is to attempt the training in a manner appropriate for that person. IQ tests should not be used to preclude training opportunities.

Adaptive behavior scales

The second part of the definition of mental retardation, quoted above, requires that subaverage intellectual func-

tioning exist concurrently with deficits in adaptive skills.⁴ Such deficits are typically measured by adaptive behavior scales; these are required to diagnose mental retardation. These scales are not typical psychological tests. Rather than presenting a task to the patient, questions are asked of a person familiar with the daily activities of the patient. An advantage of this approach is that it enables evaluation of people with severe and profound disabilities; the obvious disadvantage is that accuracy is dependent on the respondent's knowledge and ability to follow directions. The best known of these scales are published by the American Association on Mental Retardation, the AAMR Adaptive Behavior Scales⁸ and the Vineland Adaptive Behavior Scale.⁹ In both, the responses are converted into standard scores which are compared with scores obtained by people in various standardization groups, both with and without disabilities. Thus, a person's skills in a particular domain can be assessed in relation to skills of people without disabilities, in order to determine areas of deficit, and can also be compared to other people with disabilities, in similar environments, to assess training progress. Separate scores may be obtained for various "domains" such as communication, community skills, domestic skills, vocational skills, and maladaptive behavior. While IQ testing is relatively common in the general population, as well as among people with disabilities, adaptive behavior scales are primarily used with people who have mental retardation and related conditions.

There is a tendency to diagnose and classify mental retardation based upon IQ information only. However, information on adaptive behavior is more important, especially for adults. If a person can function independently in his or her environment, it makes no sense to characterize that person as having mental retardation. Conversely, a person with an IQ score above the 70 to 75 range, who is not able to function independently, certainly does have a disability.

There is some utility in repeating adaptive behavior scales at regular intervals. They provide an indicator of progress in learning new skills, especially for children and adolescents. They also reflect declines in skills which can occur as a result of aging, medication changes, illness, or an environment in which use of skills is not reinforced.

Personality and psychopathology

The psychological construct of personality is much more controversial than that of intelligence. Nevertheless, personality tests are frequently included in psychological test

batteries. The best known of the structured personality tests is the Minnesota Multiphasic Personality Inventory (MMPI),¹⁰ while the Rorschach inkblot test¹¹ is probably the most famous projective test. Neither of these, or similar instruments, can be administered to people who have serious language and communication deficits. Thus, personality testing is limited to people with mild disabilities. Due to these limitations, the vast literature on personality testing includes little with regard to mental retardation. Typically, standard personality tests are not used with people who have mental retardation; those that are used are likely to be concerned with diagnosis of personality disorders.¹² Over the past 15 years, there has been an increasing interest in measuring and treating other types of psychopathology in people with mental retardation.

Several instruments, with accumulating literatures, appear to have some utility for assessing psychopathology. The earliest and best established is the Reiss Screen for Maladaptive Behavior.¹³ As with adaptive behavior scales, the Reiss Screen is completed by a person familiar with the patient's behavior and its validity is dependent on the observer's accuracy. Also, while it serves to discriminate people with psychopathology from those without, it does not suggest a specific diagnosis. An instrument that does yield a diagnosis is the Psychopathology Instrument for Mentally Retarded Adults (PIMRA),¹⁴ but it has not been updated to use DSM-IV⁶ criteria. The Aberrant Behavior Checklist¹⁵ can be used to verify behavioral changes which may result from changes in psychotropic drugs; it was designed specifically for people with mental retardation. Aman¹⁶ has reviewed a large number of instruments available for assessing psychopathology in people with mental retardation, along with recommendations for their use.

Tests for psychopathology still have a variety of shortcomings but are improving. Despite the shortcomings, they can provide useful information relevant to treatment of people who do have a relatively high rate of behavior problems and psychotropic drug use. Thus, it is surprising they are not often used in psychological testing of people with mental retardation. As these kinds of instruments are improved, and become better known, it is likely their use will increase. There is not much that can be done about IQ scores of adults. Treatment decisions are likely to center around behavioral problems. Tests that provide information about such problems show the most potential for assessing adults.

Future trends

Many other kinds of tests are widely used in psychological practice, but not discussed in this article because they are

generally not used with adults who have mental retardation. One type which may seem especially relevant is neuropsychological testing.¹⁷ While there is research attempting to relate brain function and behavior in mental retardation, there is little literature describing clinical use of these tests. Practical problems, such as lack of skills needed to follow test directions and expense of administration, prevent widespread use. Of potentially greater interest are neuroimaging techniques which are being used to study etiologies of mental retardation.¹⁸ However, these techniques are expensive and it may be some time before they become available to many people with mental retardation.

Conclusion

Diagnosis of mental retardation must take place by the age of 18 years and requires IQ testing and assessment of adaptive functioning. IQ tests are academic and have some utility in educational placement of children. Repeating IQ tests during the adult years is not of use to people with mental retardation, nor for most adults, whether they have disabilities or not. Assessment of adaptive behavior can be useful for adults because it is expected that teaching and maintenance of life skills will continue throughout the life span. Thus, skills should be expected to improve over time, although rapid changes in adaptive behavior scores should not be expected. A decline in adaptive skills could indicate a medical problem or an inadequately stimulating environment.

Traditional personality testing is generally not done with people who have mental retardation, but methods for measuring psychopathology and personality disorders are starting to be used. Since people with mental retardation have higher rates of behavioral disorders than the general population, such instruments are potentially useful in treatment planning and evaluation.

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Morningstar risk-adjusted performance ratings for the periods ended 4/30/97**

Fund	Overall	3 yr	5 yr	10 yr
Tax-Free Short-Intermediate	★★★★★	★★★★★	★★★★★	★★★★
Tax-Free Insured Intermediate	★★★★	★★★★	—	—
Tax-Free Income	★★★★	★★★★★	★★★★	★★★
Tax-Free High Yield	★★★★★	★★★★★	★★★★★	★★★★★
California Bond	★★★	★★★★★	★★★★	★★
Florida Insured Intermediate	★★★★	★★★★	—	—
Georgia Bond	★★★★★	★★★★★	—	—
Maryland Bond	★★★★	★★★★★	★★★★	★★★★
Maryland Short	★★★★	★★★★	—	—
New Jersey Bond	★★★★	★★★★	★★★★	—
New York Bond	★★★★	★★★★	★★★★	★★★
Summit Municipal Income	★★★★★	★★★★★	—	—
Summit Municipal Intermediate	★★★★★	★★★★★	—	—
Virginia Bond	★★★★	★★★★★	★★★★	—

Virginia Short-Term Bond Fund has a performance record of less than 3 years and is therefore not rated.



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**Morningstar proprietary ratings reflect historical risk-adjusted performance through 4/30/97. These ratings are subject to change every month. Ratings are calculated from the funds' 3-, 5-, and 10-year average annual returns in excess of 90-day Treasury bill returns with appropriate fee adjustments and a risk factor that reflects fund performance below 90-day Treasury bill returns. The 1-year rating is calculated using the same methodology but is not a component of the overall rating. For the 1-year period, the following funds were rated among 1,745 municipal funds: Tax-Free Short-Intermediate received 4 stars; Tax-Free Insured Intermediate received 3 stars; Tax-Free Income received 5 stars; Tax-Free High Yield received 5 stars; California Bond received 5 stars; Florida Insured Intermediate received 3 stars; Georgia Bond received 5 stars; Maryland Bond received 5 stars; Maryland Short received 4 stars; New Jersey Bond received 5 stars; New York Bond received 4 stars; Summit Municipal Income received 5 stars; Summit Municipal Intermediate received 5 stars; and Virginia Bond received 5 stars. Ten percent of the funds in an investment category receive 5 stars, the next 22.5% receive 4, the next 35% receive 3, and the next 22.5% receive 2. †\$500 minimum.

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MARYLAND MEDICAL HISTORY

Looking back to May 1909: Opening of the new faculty building—1211 Cathedral Street

Introduction

The Medical and Chirurgical Faculty of Maryland (Med Chi) is on the verge of celebrating its 200th anniversary; it is among the oldest medical societies in the United States. At the time of Med Chi's 100th anniversary, Dr. Eugene Fauntleroy Cordell (**figure 1**) published the *Medical Annals of Maryland*, describing the history of Maryland physicians and Maryland medicine. Dr. Thomas Cullen (**figure 2**) said, "It is the bedrock on which the later medical histories of the state will be built."

In the early 20th century, there were eight medical schools in Baltimore and close to 200 in the country. Education standards were generally poor, research was relatively nonexistent, and the public was provided with improperly prepared physicians for whom there was little respect. The Johns Hopkins Medical School was founded in 1889, with the guidance of four great pioneers, Kelly, Halstead, Osler, and Welch, as well as others. The Abraham Flexner Report, commissioned by the Carnegie Foundation, was rendered in 1911. Soon two schools remained in Baltimore — The Johns Hopkins Medical School and The University of Maryland Medical School — when the Baltimore Medical College (Maryland General Hospital) and the College of Physicians and Surgeons (Mercy Hospital) merged with the University of Maryland in 1912 and 1915, respectively. Then, in 1920, after Hopkins had become nationally known, the General Assembly in Annapolis questioned the need for two medical schools in our small state. But Dr. Gordon Wilson and Dr. Arthur Cantwell of North East, Maryland, the latter then Senator from Cecil County, led support for the University of Maryland and stressed its importance in producing physicians.



Figure 1. Dr. Eugene Fauntleroy Cordell



Figure 2. Dr. Thomas Cullen



Figure 3. Medical Group at 1211 Cathedral Street
May 13, 1909 — Opening of New Faculty Building

During this exciting period, Maryland's growth and prominence in the profession was marked by the dedication of the new Med Chi building, which opened on May 13, 1909. A photograph of distinguished physicians who attended (figure 3) prompted the writing of a few mini biographies of several prominent persons shown in the foreground (figure 4). Those highlighted include Dr. William Osler, Cardinal James Gibbons, and Drs. Abraham Jacobi, Clotworthy Birnie, and William Welch.

William Osler of Baltimore, 1849-1919²⁻⁵

Dr. Osler's (figure 5) life is probably more widely known than any other American physician. He was in Baltimore from 1889 to 1904 when he organized and developed a model of clinical instruction and medical investigation for students and young physicians. His colleague, Dr. William Welch said of him:

"Osler was the best type of clinician produced in the 19th century belonging, as he did, to the lineage of Laennec, Louis, Bright, Addison, Graves, Stokes, Frerichs, Jackson, Flint, Delafield, Janeway, and Pepper. By early studies, he acquired the naturalists' habit of mind. He approached the study of disease in the true spirit of scientific inquiry and by accurate observation. Like most of those physicians who obtained the highest eminence from the past century, he entered the clinical field through the gateway of morbid anatomy in which his studies extended to comparative pathology. He belonged to that small, but attractive, group of physicians represented at all ages, who combined the broadest humanism with the best science of their day.

Nothing can improve upon that tribute. Of pertinence now, Dr. Osler was vitally interested in the significant link



Figure 4. L to R, 1st Row: William Osler, Goldsborough (Pres.),
Cardinal James Gibbons, Abraham Jacobi. 2nd Row: Hon. C.J.
Bonaparte, Clotworthy Birnie, William Welch

between the medical society and the profession. He spoke about the subject at the centennial celebration of the New Haven Medical Association on January 6, 1903. He was concerned with the need for continuation of education by the general practitioner. He regarded the practice of medicine as the most satisfying occupation and emphasized responsibility for its conduct at the highest ethical standards.

To Osler, society meetings afforded "professional cement," provided they were conducted in a friendly, informative, and social way. Here, with friendly intercourse, the society could settle trifling attitudes and reduce annoyances to a minimum. Also, the society "kept the man up to the



Figure 5. Dr. William Osler

times” and enabled him to “refurnish his mental shop with the latest wares.” He envisioned society meetings as necessary to dispense the “new pathology,” and of even greater importance, to present clinical case histories of patients illustrating rare and unusual forms of illness. Osler commented, “the society should be a school in which scholars teach each other,” and he regarded the demonstration of unusual cases from a physician’s practice as a way to achieve these objectives.

The society, he emphasized, was obligated to maintain a good library for its members, and include attractive reading rooms stacked with the important weekly journals and shelves of new books involving all disciplines of medicine. He always stressed the importance of a library to a medical community and regarded it as an educational center for the younger man to keep up his training and a place for the older practitioner to seek advice and reassurance. Osler strongly encouraged membership in local, state, and national societies for all to learn from their medical colleagues.

James Cardinal Gibbons of Baltimore, 1834-1921

Cardinal Gibbons parents came to New York from Ireland in 1830. As a young priest, ordained just after the start of the Civil War in 1861, he often celebrated mass in South Baltimore parishes at Canton and Locust Point. In his gentle way, he was kind to everyone, particularly children. He had a special talent in persuading the wealthy to contribute handsomely to the church and then use the funds for the poor. He was a popular public hero whose work and influence brought America closer to the Vatican. He worked ceaselessly to make the Roman Catholic Church acceptable to all faiths. As Baltimore's first Cardinal in 1887 he ultimately became known worldwide, particularly for his defense of labor unions. Presidents William Taft and Theodore Roosevelt sought his wise counsel and friendship. Thousands knelt in grief on Cathedral Street for his holy week funeral in 1921.

In the Basilica, there is a quiet alcove, to the left, under the organ loft. This lovely inscription, placed there, is a fitting tribute to Cardinal Gibbons.

James Cardinal Gibbons

Born July 23, 1834
Ordained Priest, June 26, 1861
Consecrated Bishop August 16, 1868
Cardinal of Holy Roman Church
Title of S. Maria in Trastevere
June 7, 1886
Died March 24, 1921

A mild and Paternal Ruler of his clergy and people
Honor and Glory of the American Episcopate
Model of Every Civic Virtue
Advocate of the Working Man
Friend of the Poor and the afflicted
He governed the See of Baltimore for forty-four years
In Justice, Peace and Mercy
Erected by his Priests, 1924

It was acknowledged that Cardinal Gibbons was revered by everyone and commanded the admiration and respect of the medical profession of Maryland because of his high social and ethical values.

Abraham Jacobi of New York, 1830-1919⁶

Born in Hartum, Germany, Dr. Abraham Jacobi (**figure 6**) graduated from Bonn in 1851. He settled in New York in 1853 and became the first to inaugurate systematic and special clinics in diseases of children. In 1898, the first ward in the United States designed primarily for the clinical teaching of pediatrics was opened at the Roosevelt Hospital and later was named the “*Jacobi Ward*.” In 1857, he lectured at the College of Physicians and Surgeons (Columbia), and in 1865, he became professor of diseases of children at the University of New York. He

resigned in 1870 to become clinical professor of diseases of children at the College of Physicians and Surgeons, where he remained. He was active on the staff of Mt. Sinai Hospital beginning in 1860, the Bellevue Hospital in 1873, and he was consulting physician at the Babies Hospital, the Skin and Cancer Hospital, the Orthopedic Hospital, and the Hackensack Hospital. Dr. Jacobi was more than a teacher, practitioner, hospital physician, society worker, and author. He took active interest in what was best for medi-

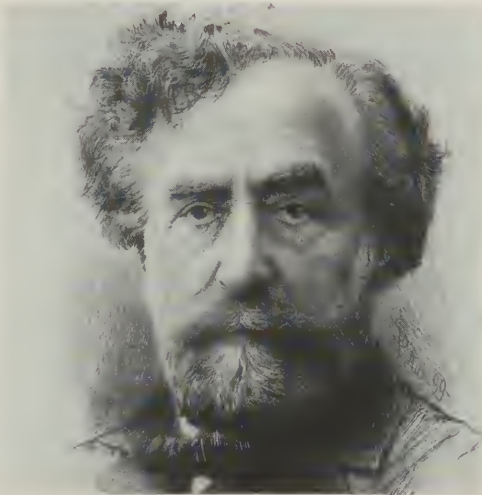


Figure 6. Dr. Abraham Jacobi

cine and served as an active public-spirited citizen. In all endeavors, he was a leader, never content to be a servile follower.

Dr. William Welch said of him:

"I consider Dr. Jacobi one of the greatest ornaments of the medical profession in this country. He has performed an essential service not only in New York, but for the profession of the whole country. Dr. Abraham Jacobi is unquestionably the Father of American Pediatrics, who has contributed more than any other person by placing pediatrics upon a firm and enduring basis."

During the very troublesome time of Bovine tuberculosis in children, Dr. Jacobi warned mothers before pasteurization was adopted: "don't forget to boil the milk."

Clotworthy Birnie of Taneytown, Carroll County, 1843 - 1917

Clotworthy Birnie (figure 7), son of Rogers Birnie and Amelia Knode Harry, and grandson of Clotworthy Birnie, Sr., was born at "Glenburn," near Taneytown, on January 13, 1843. He was the great-grandson of Upton Scott, a founder and the first president of the Medical and Chirurgical Faculty of Maryland. He studied medicine with Drs. John Swope and W. Chew Van Bibber. Dr. Birnie received his medical degree from the University of Pennsylvania in 1870. He began practice at Glenburn in 1870, but he soon relocated to Taneytown. He remained in practice for 47 years. In addition to his active professional life, Dr. Birnie supervised an active farm. After his father's death in 1891, Mrs. Birnie, Sr., lived with Dr. Birnie until her death, aged 86. Dr. Birnie never married.

He was active in politics and served as a member of the Maryland House of Delegates in 1896, and chairman of the Ways and Means Committee. Late in the nineteenth century, he was elected president of the Carroll County Medical Society. Dr. Birnie was among the first to propose the formation of a medical society for Carroll County. He was ordained an elder in the Taneytown Presbyterian Church in 1864, and was elected first president of the Taneytown



Figure 7. Dr. Clotworthy Birnie

Volunteer Fire Company in 1867. From 1895 to 1896, he was vice president of the Medical and Chirurgical Faculty of Maryland and president of the faculty from 1899 to 1900. He died of a cerebral hemorrhage on March 16, 1917, at Glenburn. He was buried at Piney Creek Presbyterian Church near Taneytown. Dr. Birnie was one of Carroll County's foremost physicians; his life was dedicated to rendering service to others regardless of reward.

In 1895, during the Ninety-Second Annual Session of the Medical and Chirurgical Faculty of Maryland on April 23, the general topic for discus-

sion was typhoid fever in county districts. William Osler read a paper and Dr. Birnie was a discussant and co-referee for the meeting. During a fall meeting at Hagerstown in 1896, Dr. Birnie presented a paper, along with sixteen others, which included distinguished physicians such as Randolph Winslow, John Hemmeter, J. Mason Hundley, William Osler, Simon Flexner and J. M. T. Finney. During his Med Chi presidency, he served as a member of the board of trustees, which included Drs. Osler, Tiffany, Welch, and Ashby.

It is of considerable interest that Dr. William Osler, Regius Professor of Medicine, Oxford, complimented Clotworthy Birnie in an address he gave to the Royal Medical Society of Edinburgh on February 2, 1907 (published in March 1907). In speaking of relations between the Royal Medical Society and the professions of the United States and Canada, Osler referred to Dr. Clotworthy Birnie and said: "A country practitioner of Maryland, whom to know, makes one proud of his profession and who could sit among you here tonight looking more like a Scot than many I see."

In the manuscript collection of the Historical Society of Carroll County, there are a number of Birnie papers relating to their farm business operations. In addition, the journals of the Birnie family have been microfilmed as part of the collection of the Maryland Hall of Records. The Birnie archival materials offer considerable insight to the early history of Taneytown.

William H. Welch of Baltimore, 1850-1934⁸⁻¹⁰

Dr. William H. Welch (figure 8) was a native of Norfolk, Connecticut.



Figure 8. Dr. William H. Welch

After graduating from Yale as a young man, he studied chemistry at Sheffield Scientific School. In 1872, he entered the College of Physicians and Surgeons and excelled as a student leader. After graduation in February 1875, he was appointed intern at Bellevue Hospital, where he was influenced by Delafield, a renowned pathologist. Disinterested in medical practice, and with his father's support, he sailed for Europe in April 1876, where he learned of experimental pathology. With Cohnheim in Germany, he studied pulmonary disorders and wound infections. Thereafter, he served successively as professor of pathology at Bellevue Hospital Medical College in New York from 1879 to 1894 and the Johns Hopkins Hospital and Medical School beginning in 1884. The Hopkins Hospital officially opened in 1889 and the Medical School in 1893.

During his chairmanships, he trained and nurtured a long line of worthy pupils who later distinguished themselves. Among them were William Councilman, Walter Reed, and James Carroll, whose later work clarified the yellow fever problem. It was Welch who, in 1900, suggested to Walter Reed "do not forget to filter it," based on his knowledge of Loeffler and Frosch's work on foot and mouth disease in cattle caused by a filterable virus.

Many contributions were made by this pioneer pathologist and investigator, including acute lung edema (1877), the staphylococcal etiology of wound infections (1892), and etiology of gas gangrene (1892). He helped clarify the mechanisms of thrombosis and embolism, and with Flexner and Von Behring, determined the action of diphtheria toxin (1891 to 1892).

A wise and forceful medical and educational statesman, Dr. Welch rendered invaluable services to various Army Surgeons General from Sternberg to Ireland and "exerted a formative influence upon military preventive medicine, a force of global dimensions."¹⁰

Dr. Welch's twilight years were devoted as the first chairman of a new department of medical history at Hopkins. The Medical and Chirurgical Faculty of Maryland was continually enriched by his wise advice and numerous educational contributions.

Conclusion

Junior and senior medical students enthusiastically attended Med Chi's monthly and annual meetings. Professors at Hopkins and Maryland often spoke, as well as invited out-of-town educators. Drs. Soma-Weiss, Samuel Levine, Paul D. White, Elliott Joslin, Chester Keefer, all from Boston, Dr. Tinsley Harrison of Alabama, Dr. Russell Cecil of New York, and Dr. Francis Wood of Philadelphia were among them.

The Medical and Chirurgical Faculty of Maryland was an important part of young medical life and development. Those who settled in Baltimore and Maryland usually became mem-

bers, and followed with membership in the American Medical Association.

As the society approaches its second millennium, we should be constantly reminded of our heritage. Dr. Thomas Cullen, in an address to the faculty in 1927, remarked:

"The history of medicine is to the practice of medicine what the foliage is to the flower; it gives it the most delightful setting. It enables us to see the hardships of the past and enables us to understand how those rugged men of honesty conquered their many obstacles. It makes better persons of us."

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Acknowledgment

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THEODORE E. WOODWARD, M.D., MACP

Dr. Woodward is professor of medicine emeritus, department of medicine, University of Maryland School of Medicine and the Baltimore Veterans Administration Medical Center. Dr. Woodward was formerly professor and chairman, department of medicine, University of Maryland School of Medicine, and physician-in-chief, University Hospital, Baltimore. ■

Reprints: Theodore W. Woodward, M.D., 1 Merrymount Rd., Baltimore, MD 21210

FROM THE BPQA

A crime of moral turpitude

The Internal Revenue Service (IRS) notified the Board of Physician Quality Assurance (BPQA) that one of our licensees had been found guilty of attempting to bribe an IRS agent. Penalties included a suspended jail sentence, probation, forfeiture of the bribe money, extensive community service, and notification of the BPQA.

■ If you were a member of the BPQA, what would you do with this physician?

Regardless of what you would like to do, the law specifies that if a physician is found guilty of, or pleads guilty to, a "crime of moral turpitude," the BPQA is required to revoke his or her medical license. This physician was charged under this provision of the Maryland Medical Practice Act. The physician requested a Case Resolution Conference at which he admitted he had done something very foolish. He explained that bribery of officials was common in his country of origin. No allegations had been made about his standard of medical practice. In fact, the judge's order suggested the community service requirement might be satisfied through the provision of free medical services.

■ If you were a member of the BPQA, would this further information affect your decision on how to resolve this case?

The members of the Case Resolution Conference suggested an innovative resolution. Typically, when a physician's license is revoked, he or she is out of prac-

tice at least 12 months. Since a physician cannot practice medicine without a license, revoking the license would preclude this physician from performing community service by providing free medical care. Because the BPQA members felt this was the most valuable and meaningful retribution the physician could provide to fulfill the court order, they reasoned that the law could be satisfied by restricting the physician's license for one year so that only free services could be performed. The physician would be allowed to retain sufficient fees to cover malpractice insurance and to pay support staff. This recommendation was referred to the whole BPQA for its decision.

■ If you were a BPQA member, would you accept this recommendation, or would you support the usual course of revocation for a minimum of 12 months?

The BPQA felt the public would be better served by 12 months of free care than by 12 months of "down time." Because the crime did not involve the practice of medicine, and because the board accepted the explanation that the physician had acted in a manner consistent with practices tacitly accepted in his country of origin, there was a consensus that restricted licensure was an option.

Comment by Dr. Cheryl Winchell, a member of the BPQA:

This case was unique. If Medicare or Medicaid fraud had been involved, this physician would have received a simple revocation. Board members were flab-

bergasted that anyone would be foolish enough to try to bribe an IRS agent!

In my five years serving on BPQA, I have been impressed with the disproportionate number of foreign trained physicians who come to the board's attention. International medical graduates (IMGs) are three times more likely to be disciplined than American-trained physicians. **This is not the result of any bias of the BPQA. The board is geared to respond to complaints made by others. Often, disciplinary actions are initiated without the BPQA being aware of the physician's gender, let alone his or her nationality or the fact that he or she graduated from a foreign medical school.** It seems clear that cultural factors cause the disproportionate representation of IMGs among our disciplines.

Most physicians whose licenses have been revoked for Medicare and Medicaid fraud are IMGs. They also are disproportionately represented in cases involving sexual violation of patients. Perhaps doing a favor for a friend by providing a bogus medical excuse for illness, altering a medical document, or prescribing for someone without having a *bona fide* doctor-patient relationship are all behaviors which may be accepted in other cultures. IMGs may not share the board's concerns about the boundaries between physician and patient and may feel the BPQA's actions are both unfair and unreasonable. But, immigrants must appreciate that they will be judged by our ethic, and not the ethic of their native country.

The BPQA intends to initiate an orientation program for new licensees which will apprise them of the details of the Medical Practice Act and other laws that apply to the practice of medicine in Maryland. We will also attempt to point out the cultural differences that have led to physician discipline. Additionally, the board has been providing speakers for medical staff and medical society functions on request. We feel the public is best protected by the BPQA acting to educate physicians and hopefully prevent problems that may lead to discipline. ■

Books, Etc.

Book review editor:
Chris Papadopoulos, M.D.

How Physicians Can Avoid Surrender and Lead Change.

Elizabeth Gallup, M.D., J.D., MBA, with Cyd Slayton. Tampa, FL: American College of Physician Executives (ACPE); 1996. 89 pages.
\$38.00 for non-ACPE members (paperback).

The book's title and subtitle, "Gaining Real Influence in Your Own Health Care Before it's Too Late," clearly indicate what the authors hope to convey to the readers.

Dr. Gallup, the primary author, is a board-certified family physician, a licensed attorney, and has a master's in business administration. She has been a practicing physician, a faculty member of a medical school, and a vice president for business development of a hospital. She is currently executive director of a physician service network (PSN) in Kansas City. Her co-author is vice president of a health consulting organization and has served as a marketing, communications, and management counsel to hospitals, physician groups, and managed care organizations.

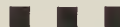
The volume is a 101 course on business methods as they apply to physicians in today's environment. It is also a primer on human relations and mass psychology. The authors divide the book into 10 chapters, each devoted to one aspect of the theme. Within each of the chapters are numerous focused headlines concentrating on specific points within that topic. The discussion of each utilizes anywhere from a quarter page to a number of pages.

Much of what is presented is common sense. However, often when one is so closely, emotionally, and vitally involved in a matter, as we physicians are, one "fails to see the forest for the trees."

While the authors once refer tangentially to the Federal Trade Commission's more recently "altering the anti-trust climate" based on "the rule of reason," they don't mention the commission's threat of prosecution under anti-trust laws for physicians who formed their own HMOs. They also leave the impression that HMOs are the final state in our search for a more efficient, less costly medical care program. The facts that supply will never equal demand, changes need to occur inpatient expectations, tort reform is essential, and defensive medicine can no longer be tolerated are not discussed. They fail to contemplate that no one can foresee the future costs of yet-to-be discovered tests and therapies, although they do comment on the comparative greater expenditures by the public for entertainment than for medical care.

The volume reads easily and quickly. It will help physicians better organize their thoughts on these vital issues.

MARION FRIEDMAN, M.D. ■



Genesis. A Living Conversation. Bill Moyers. New York: Doubleday; 1996. \$29.95.

In *Genesis, A Living Conversation*, forty-eight conversationalists discuss the book of Genesis with Bill Moyers, the group leader. Each of these individuals, a dazzling array of writers,

theologians, artists, and thinkers, has made a significant contribution to the text.

Moyers experienced an extraordinary career in theology, journalism, govern-

The CMERC Update, now in its second year, informs all Med Chi accredited CME sponsors about the activities of the Continuing Medical Education Review Committee (CMERC). The CMERC has received feedback (both negative and positive) from our accredited sponsors this past year. This exchange of information has kept us all better informed.

CMERC workshop a success

The Continuing Medical Education Review Committee's (CMERC) workshop, held during the Med Chi annual meeting on May 2, 1997, at the Turf Valley Inn and Country Club in Ellicott City, was a big success.

The workshop was attended primarily by representatives of Med Chi accredited continuing medical education sponsors in Maryland. Ninety-eight percent of the attendees rated the workshop overall excellent to good. Participants offered many useful suggestions to consider for future workshops.

The CMERC extends its thanks and appreciation for the time, effort, and support of its members and staff in making this meeting a success. David Solomon, M.D., a CMERC member, discussed the committee's new *Guidelines and Policies on Mergers, Acquisitions, or Other Organizational Restructuring Involving Med Chi Accredited Institutions*. Deusdedit L. Jolbitado, M.D., gave a brief summary of the activities of the CMERC from April 1996 to April 1997.

The committee would especially like to offer sincere thanks to those invited speakers from our accredited CME sponsors. Clifford G. Andrew, M.D., and Alison Rolon of Anne Arundel Medical Center both gave excellent presentations on "Ensuring and Documenting Linkage in Essentials #2 through #5." Richard A. Farson, M.D., and Laura Austin of Southern Maryland Hospital Center offered practical ideas and methods for documenting conversations relating to CME activities. Mindel Shore, LCSW-C, of CPC Health, Inc., also gave an excellent presentation on the benefits and problems encountered, and solutions found, during the recent merger of the CME committees of two accredited organizations.

Med Chi board of trustees approves CMERC policy

The CMERC's *Guidelines and Policies on Mergers, Acquisitions, or Other Organizational Restructuring Involving Med Chi Accredited Institutions*, which was approved by the CMERC at its meeting March 19, 1997, and discussed by David Solomon, M.D., at the recent CMERC workshop, was approved by the Med Chi board of trustees at the April 17, 1997, meeting. Copies of this policy will soon be sent to all Med Chi accredited sponsors.

Reminder to all intrastate accredited CME sponsors

Med Chi is the accrediting body for intrastate CME sponsors in Maryland. It is not appropriate for an intrastate sponsor to regularly offer CME activities which are regional or national in scope (i.e., advertised to physicians outside of Maryland or bordering states). If a Med Chi accredited CME sponsor is considering planning a CME activity that is regional or national in scope (intrastate sponsors may do this once per year with Med Chi's permission), that sponsor should obtain permission from the CMERC before the planning is started. If a CME sponsor is considering offering CME activities which are regional or national in scope more frequently than once a year, that sponsor should seek national accreditation from the ACCME (Accreditation Council for Continuing Medical Education).

CME sponsors' feedback on the new CMERC handbook

The CMERC has received very positive feedback on the *CMERC Handbook* that was sent to all Med Chi accredited CME sponsors last year. In addition to feedback that has been given informally over the past year, comments were invited on the evaluation form that was distributed at the

recent workshop. Some of the comments that we received included:

"The *CMERC Handbook* is the most useful tool the committee has produced."

"[The handbook] will be helpful to the sponsors in accreditation surveys."

"Very helpful to a new coordinator."

"The examples were especially handy along with the tabs directing us to specific needs."

"Excellent! I use it frequently. Thank you."

Some workshop participants commented that they would like to have additional copies of the handbook for other staff or committee members. Accredited sponsors should feel free to make as many copies as they wish for use within their own programs.

News alert for CME sponsors

An article in the May 12, 1997, issue of *AMA News* should alert CME sponsors to a very controversial court decision which could seriously impact on medical meetings in the future. Four national medical specialty societies are being sued because their educational meetings are claimed to be "illegally promoting a device." A United States District Court judge in Philadelphia decided not to dismiss the lawsuits.

DEUSDEDIT L. JOLBITADO, M.D.

Dr. Jolbitado is chairperson of Med Chi's Continuing Medical Education Review Committee ■

The purpose of this newsletter is to inform CME chairpersons, CME committee members, and all interested physicians about the activities of Med Chi's Continuing Medical Education Review Committee (CMERC) and about the rules and procedures that affect the implementation of CME programs in Maryland. When appropriate, news from the ACCME (Accreditation Council for Continuing Medical Education) is included.

Med Chi Bicentennial Celebrations

*Med Chi has already begun planning celebration activities
for its bicentennial in 1999.*

*If you have ideas or suggestions, please call Margaret Burri at
410-539-0872 or 1-800-492-1056.*



EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

July, 1997

Selected Communicable Diseases in Maryland in 1996

(Continued from May/June, 1997)

Please refer to the May/June issue for a description of communicable disease surveillance in Maryland and for Tables 1a and 1b, "Cases of Selected Notifiable Disease Reported in Maryland in 1996 by County". All incidence rates in this article are expressed as cases per 100,000 population per year. All rate maps place cases by county of residence at the time of diagnosis. The spot maps place cases within their zipcode of residence, but with county boundaries displayed on those maps.

HEPATITIS A (256)

5.0/100,000 (U.S. 10.9/100,000)

The trend in hepatitis A from 1987 to 1996 is illustrated in Figure 4. The incidence rate in 1996 (5.0) increased slightly from the rate in 1995 (4.4). In 1996, Montgomery County, Prince George's County and Baltimore City accounted for 55% of the cases in the state with 54, 44, and 41 cases respectively. The highest rate occurred in Dorchester County (13.2), followed by Calvert (7.6), Harford (7.5%), St Mary's (7.3). The Baltimore City rate fell 38%, from 9.5 in 1995 to 5.9 in 1996 (Figure 5).

The ratio of male to female cases was 1.9:1. The case ratio of whites to non-whites was 2.2:1; the rate in whites was 4.6 compared to 4.9 in non-whites. Non-white males had the highest rate (7.6) followed by white males (5.9) and white females (3.6).

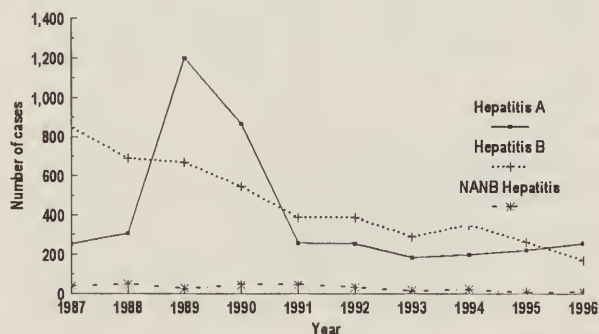


Figure 4. Hepatitis (A, B, and NANB).
Cases reported, Maryland, 1987-1996.

Of the 195 people reporting an occupation, 24 reported jobs with increased risk of acquiring or transmitting hepatitis A: 14 food handlers, 9 health care providers, and 1 child care provider. Two children attended day care.

Among the 243 (95%) cases on whom information was available, 51 (21%) had contact to a confirmed or suspected case of hepatitis A, 31 (13%) had travel outside the U.S. or Canada, 25 (10%) had consumption of raw shellfish, and 15 (6%) were suspected of being part of a common-source outbreak.

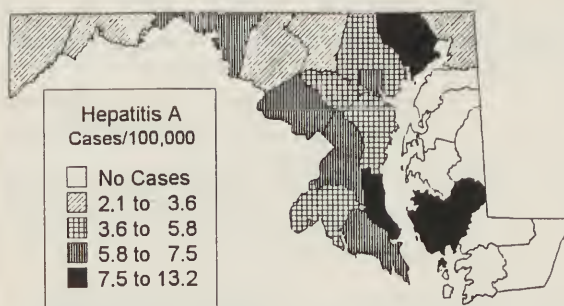


Figure 5. Hepatitis A. Incidence in Maryland, 1996.

HEPATITIS B (169) 3.3/100,000 (U.S. 3.8/100,000)

The trend for hepatitis B from 1987 to 1996 is illustrated in Figure 4. This year we performed quality control measures to ensure that all cases strictly met the CDC case definition for acute hepatitis B. The incidence rate in 1996 (3.3) decreased from the rate in 1995 (5.2). A large part of the decline may be attributed to stricter adherence to the CDC case definition and exclusion of reported cases that did not meet the definition. (A total of 41 additional suspect cases that were HB_eIgM positive were excluded because they were missing information about jaundice, elevated liver function tests, or acute onset of disease.) The highest rate occurred in Charles County (8.7), followed by Garrett (6.7), and Prince George's county (5.3) (Figure 6).

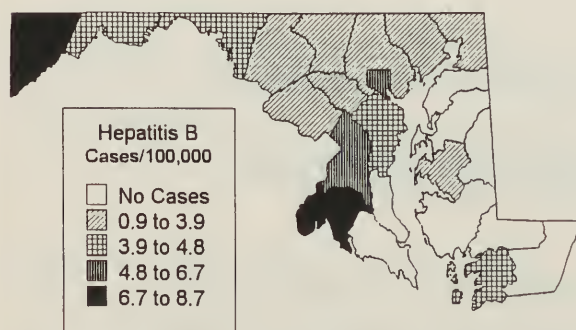


Figure 6. Hepatitis B. Incidence in Maryland, 1996.

The ratio of male to female cases was 1.2:1. The incidence rate was 3.8 in males and 2.9 in females. Overall, the incidence rate was highest in the 20 - 29 years age group (8.6). Case rates by age

group and sex appear in Figure 7. Among the 95% of cases for which race is known, the rate in non-whites (4.2) was nearly 3 times higher than the rate in whites (1.5).

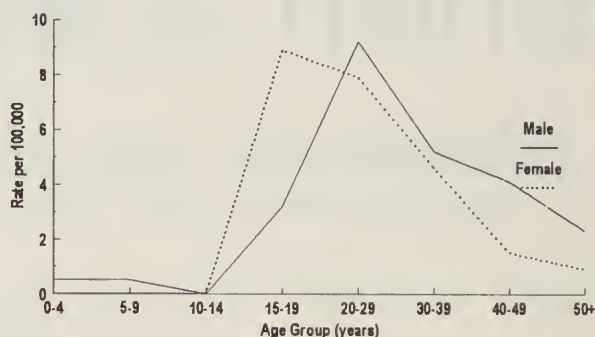


Figure 7. Hepatitis B. Incidence by age group and sex, Maryland, 1996.

Of the 93 adults with known occupation (excluding students and unemployed), only 5 were health care providers with direct patient contact.

Information on exposures with potential risk for acquiring hepatitis B during the 6 months prior to onset of illness was available for 86 (51%) of the cases. Sixty-four reported a single risk factor while 22 reported multiple risk factors. The risk factors reported included multiple sexual partners (27), contact with a confirmed or suspected case of hepatitis B (23), dental work or oral surgery (17), use of needles for injection of street drugs (11), homosexual or bisexual preference (8), surgery (7), medical or dental field employee (5), and transfusion (4).

LEGIONELLOSIS (39) 0.76/100,000 (U.S. 0.4/100,000)

The 39 reported cases of legionellosis in 1996 represented a 34% increase over the number of cases in 1995 (29). The number of cases by jurisdiction is shown in Table 1b. Dorchester County (1 case) had the highest rate (3.3) in the state.

The male:female ratio was 1.4:1. The incidence rate in females (0.6) was lower than the rate found in males (0.9). The ratio of white to non-white cases was 3.2:1. Ages ranged from 25 to 93 years (median 57 years). The incidence by age group and

sex is shown in Figure 8. There were 5 deaths among 38 cases with known outcomes yielding a case fatality rate of 13.2%.

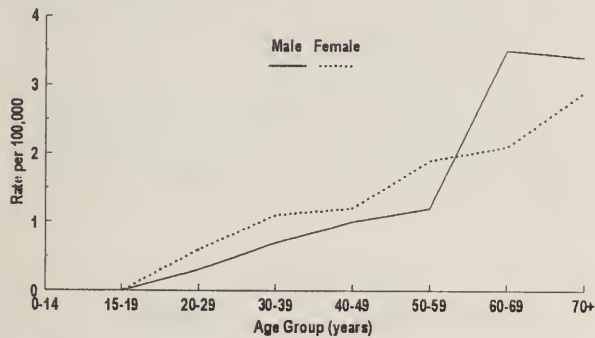


Figure 8. Legionellosis. Incidence by age group and sex, Maryland, 1996.

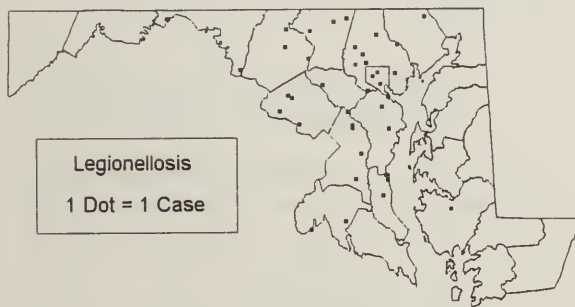


Figure 9. Legionellosis. Maryland, 1996.

Of the 21 cases on whom information was available, 3 (14.3%) were smokers of more than ten cigarettes a day. None of the three smokers reported other predisposing factors. Among the non-smokers, one was receiving treatment with immunosuppressive drugs, one individual had diabetes, one individual was diagnosed with cancer, and two individuals were diagnosed with systemic lupus. The 21 cases noted these possible exposure sources: hospital stays (2), hospital outpatient visits (3), and overnight travel (3).

LYME DISEASE (423) 8.3/100,000 (U.S. 5.2/100,000)

During 1996, 448 cases of Lyme disease were reported, 25 of whom had an onset prior to 1996. This report summarizes the remaining 423 cases

whose onset was in 1996. Lyme disease increased by 26% from 1995 (336 cases). The trend over the past ten years is shown in Figure 10.

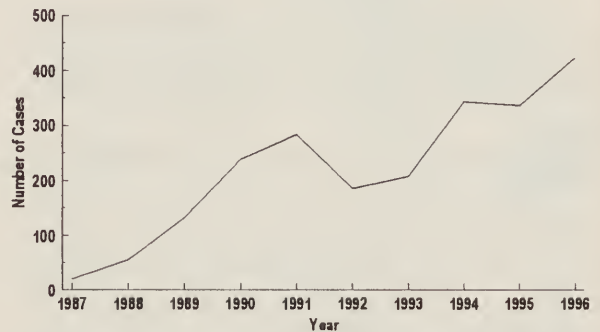


Figure 10. Lyme disease. Cases reported, Maryland, 1987-1996.

The highest rate occurred in Kent County (111.8), followed by Queen Anne's (60.3), Talbot (33.6), and Calvert (33.1) counties (Figure 12).

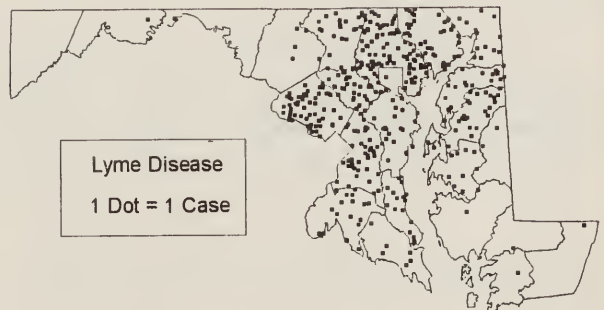


Figure 11. Lyme disease, Maryland, 1996.

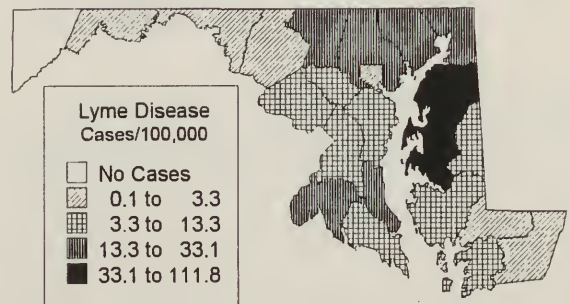


Figure 12. Lyme disease. Incidence, Maryland, 1996.

The male to female ratio was 1.2:1. Among the 384 cases with known race, 365 (95.0%) were white, 13 (3.4%) were black and 6 (1.6%) were other races. Ages ranged from 1 to 90 years (median 37 years). Thirty-three percent of the patients were under 20 years of age, as was the case in 1995.

A definite tick bite prior to onset was reported in 166 (39%) cases, 199 (47%) had no known tick exposure, and 58 (14%) were uncertain of exposure.

Eighty-five percent of the 423 cases had onsets of illness in May through September; the peak incidence was in June (137 cases). Of the 423 cases, 286 (68%) had erythema migrans, 86 (20%) had arthritis, 62 (15%) had Bell's palsy, 15 (4%) had lymphocytic meningitis, 7 (2%) had radiculoneuropathy, 5 (1.2%) had atrioventricular block, and 3 (0.7%) had encephalitis.

MALARIA (87)

1.7/100,000 (U.S. 0.6/100,000)

Eighty-seven cases of malaria were reported in 1996 compared to 63 cases in 1995. All cases were laboratory confirmed.

Seven counties and Baltimore City reported malaria cases in 1996. The majority of cases occurred in Prince George's (44.8%) and Montgomery (37.9%) Counties.

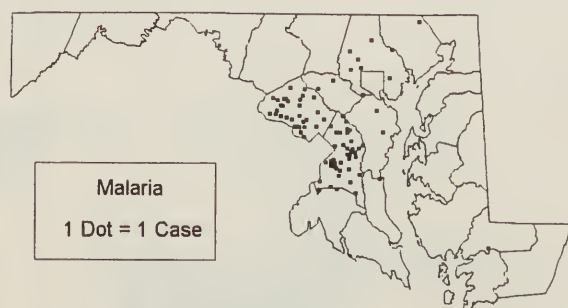


Figure 13. Malaria (all species), Maryland, 1996.

Cases were seen almost 6 times more frequently in non-whites than whites. The male to female ratio was 1.3:1. Cases ranged in age from 10 months to 72 years (median age 33). Thirty-seven (42.5%) were hospitalized; no deaths were reported. Of the 68 cases for which previous malaria status is known, 54% reported previous episodes.

Two species accounted for most of the cases: *P. falciparum* 44 (50.%) and *P. vivax* 16 (18.4%). There were 6 cases of *P. malariae* and 4 cases of *P.*

ovale. The species was unknown for 17 cases. One case had two species identified on blood smear.

Of the 87 cases, 85 (97.7%) were judged to be imported; information for the remaining two was unavailable. Overall, 22 (25%) of all cases were acquired from Nigeria. *P. vivax* was acquired from India and West Africa. *P. falciparum* and *P. ovale* was acquired primarily from west Africa, *P. malariae* from Nigeria and Ghana. Of the 69 cases for which information on prophylaxis was available, only 17 (25%) reported taking any prophylaxis prior to their illness.

MEASLES (2)

0.02/100,000 (U.S. 0.2/100,000)

Two cases of measles is the second lowest number of cases recorded in Maryland since records have been kept. Both cases were international importations, so indigenous transmission of measles in Maryland has been interrupted for nearly 24 months. The prior 5 year median (1991-1995) is 17 cases of measles. The trend in measles 16.

MENINGITIS (ASEPTIC) (210)

4.1/100,000 (U.S. - N/A)

The number of cases reported declined by 35.4%, from 325 cases in 1995 to 210 cases in 1996. There were 4 deaths among the 325 cases resulting in a case fatality rate of 1.9%. The two counties which had the highest number of cases were Montgomery (48) and Prince George's (43). The highest incidence rate (18.0) was observed in Washington County which reported 23 cases. More than half (54.8%) of the cases occurred between July and October.

The 1996 incidence rate for males was 4.3, slightly higher than the rate of 3.9 for females. The rate for whites was 3.9 compared to a rate of 3.6 for non-whites. (Race was unknown for 14 (6.7%) of the cases.) Age-related incidence was highest (16.1) in the birth to 4 years age group; children under 1 year of age accounted for 52 (86.7%) of the 60 cases in this age group. The etiology was reported for only 6 cases: 4 enterovirus and 2 cytomegalovirus.

MENINGOCOCCAL DISEASE (58)

1.1/100,000 (U.S. 1.2/100,000)

The number of reported cases increased by 38%, from 42 cases reported in 1995 to 58 cases in 1996. Table 1a shows the number of cases by jurisdiction. Baltimore City (13), Baltimore

County (10), Prince George's County (9), and Anne Arundel County (8) accounted for 69.0% of the 1996 cases. More than half of the cases (51.7%) occurred between March and June.

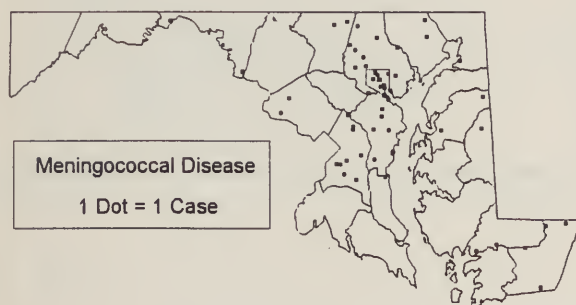


Figure 14. Meningococcal disease (all serogroups), Maryland, 1996.

The case rate for non-white races (1.8) was about twice the rate for whites (0.8). Three age groups were notable for their high incidence rates: birth to 4 years (4.3), 15-19 years (4.7), and 80 years and over (4.0). These three age groups accounted for 60.3% of all cases. Twenty-seven of the cases presented with meningitis, 23 with meningococcemia, 6 with pneumonia, 1 with arthritis, and 1 with endocarditis. There were 6 deaths among the 58 cases, for a case fatality rate of 10.3%; in 1995 the case fatality rate was 9.5%.

MUMPS (41)

0.8/100,000 (U.S. 0.3/100,000)

The 1996 case total was the third lowest total on record since record keeping began in 1920, continuing a downward trend in mumps incidence since 1991. No cases were reported from 10 (42%) of 24 jurisdictions. Of the 29 cases where laboratory results were known, 9 (31%) were laboratory confirmed. The prior 5 year median (1991-95) is 84 cases per year. The trend in mumps incidence rates over the last 10 years is shown in Figure 16.

PERTUSSIS (278)

5.5/100,000 (U.S. 2.4/100,000)

The number of reported cases increased nearly five-fold from 49 cases reported in 1995 to 278 cases reported in 1996. The trend over the past 10 years is shown in Figure 16. Cases were reported in 19 counties and Baltimore City. Cases in Frederick, Howard, and Anne Arundel Counties, and Baltimore City accounted for over half the cases statewide (151, 54.3%). The

number of cases by jurisdiction is shown in Table 1a. There were outbreaks in Howard and Frederick Counties.

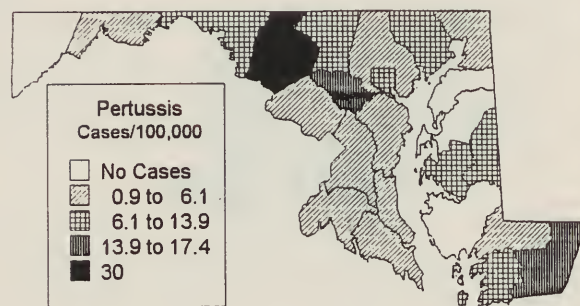


Figure 15. Pertussis. Maryland, 1996.

The male to female ratio was 0.8:1. The ratio of white to non-white cases was 4.0:1. Ages ranged from 10 days to 71 years. The median age was seven years. Seventy-six cases (27%) were less than one year old, including six (2%) newborns and 58 (21%) who were one to five months old.

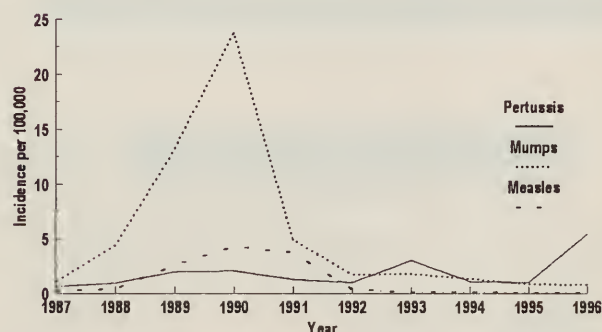
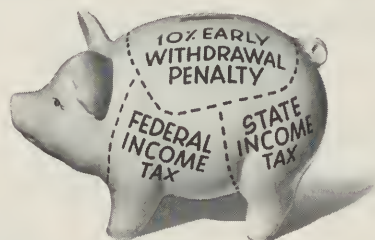


Figure 16. Measles, mumps, and pertussis. Incidence, Maryland, 1987- 1996.

The following symptoms were reported: paroxysmal cough (87%); post-tussive vomiting (56%); whoop (44 %); and apnea (26%). No deaths were reported.

Only 171 (61.5%) of the cases were cultured; 50 cases (30%) were culture positive, of which 20 (41%) were also DFA positive.

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All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

- | | |
|---|--------------------|
| Fifth annual advanced topics in CT with emphasis on spiral CT , sponsored by the department of radiology, at the Eldorado Hotel, Sante Fe, New Mexico. Credits: 16 Cat 1 AMA credits. Fee: \$525/physicians; \$475/residents, fellows. | July 24-27 |
| 25th annual geriatrics symposium: current topics in geriatrics , sponsored by the Johns Hopkins Geriatrics Center, at the Renaissance Harborplace Hotel, Baltimore. Credits: 19 Cat 1 AMA credits. Fee: \$400/physicians; \$300/residents, fellows, allied health professionals. | Aug. 21-23 |
| 26th annual diagnostic ultrasound in gynecology and obstetrics and abdomen , sponsored by the Johns Hopkins department of radiology and radiological science, at the Renaissance Harborplace Hotel, Baltimore. Credits: TBD. | Sept. 5-7 |
| Fifth annual progress in hematologic malignancies and bone marrow transplantation , sponsored by the division of hematologic malignancies, department of oncology, Johns Hopkins. Credits: 7.5 Cat 1 AMA credits. Fee: \$100/alumni, past registrants; \$125/new registrants. | Sept. 19 |
| 23rd annual topics in gastroenterology and liver disease , sponsored by the Meyerhoff Center for Digestive Disease. Credits: 24 Cat 1 AMA credits. Fee: \$535/physicians; \$285/residents, fellows. If postmarked prior to August 1: \$495/physicians; \$250/residents, fellows. | Sept. 24-26 |
| 39th annual Emil Novak memorial course: gynecology, gynecological pathology, endocrinology, and high risk obstetrics , sponsored by the Johns Hopkins department of obstetrics and gynecology, at the Hyatt Regency Inner Harbor Hotel, Baltimore. Credits: 45.5 Cat 1 AMA credits. Fee: \$950/physicians; \$750/residents, fellows. | Oct. 5-10 |

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Self-Directed CME Activities

Disease management of lipid disorders (audio tape and test). Credit: 1 Cat 2 AMA credit. Expires 6/97.

CD-ROM-based interactive multimedia radiology teaching file for Mac or PC w/single user licenses (SUL), site licenses (SL), or multisite licenses (MSL). Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Clinical breast examination using MammaCare technique: a practicum for physicians, sponsored by the Medical and Chirurgical Faculty of Maryland, at Carroll County General Hospital, Westminster, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	July 23
Cancer prevention in community practice, sponsored by the Medical and Chirurgical Faculty of Maryland, at Franklin Square Hospital Center, Baltimore County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.	July 24
Imaging in Santa Fe, sponsored by the American Association of Physician Specialists and the International Institute for Continuing Medical Education, at the Eldorado Hotel, Santa Fe, New Mexico. Credits: 25 Cat 1 AMA credits pending . Fee: \$625/physicians; \$425/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	July 28–Aug. 1
Clinical breast examination using MammaCare technique: a practicum for physicians, sponsored by the Medical and Chirurgical Faculty of Maryland, at Union Hospital, Elkton, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	Aug. 20
Neuroradiology review: including the head, neck and spine, sponsored by the University of California, Irvine, at The Four Seasons Hotel, Newport Beach, CA. Credits: 28 Cat 1 AMA credits. Fee: \$725/physicians; \$450/residents, fellows, technologists, retired physicians. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	Aug. 31–Sept. 4
Clinical breast examination using MammaCare technique: a practicum for physicians, sponsored by the Medical and Chirurgical Faculty of Maryland, at Kent and Queen Anne's Hospital, Chestertown, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	Sept. 3
The International Skeletal Society 24th annual refresher course, sponsored by The International Skeletal Society, at The Sweeney Convention Center, Santa Fe, New Mexico. Credits: 24.5 Cat 1 AMA credits. Fee: \$650/physicians; \$425/residents, fellows, technologists, military personnel, retired physicians. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	Sept. 10–13

Miscellaneous (continued)

- Mount Sinai 1997 update: brain, spine, neurovascular & ENT imaging**, sponsored by The American Association of Physician Specialists, and The International Institute for Continuing Medical Education, at The Plaza Hotel, New York, New York. Credits: 25.5 Cat I AMA credits. Fee: \$675/physicians; \$375/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Sept. 10-14**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Carroll County General Hospital. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Sept. 12**
- Organ imaging review 1997**, sponsored by The University of Toronto, department of medical imaging, at The Toronto Hilton, Toronto, Ontario, Canada. Credits: 28 Cat I AMA credits. Fee: \$520/physicians; \$370/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Sept. 14-18**
- Clinical breast examination using MammaCare technique: a practicum for physicians**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Peninsula Regional Medical Center, Salisbury, MD. Credits: 2 Cat I AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056. **Sept. 19**



PHYSICIAN'S RECOGNITION AWARD

During April 1997, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

Jeffrey Alan Abend	Naomi Ihedioha	Vernon M. Smith
Ruben F. Ballesteros	S. David Krimins	Neil Spiegel
Paul David Barnes	Leela Krishnamurthy	Richard Malcolm Susel
Lisa Ann Black	Michael Fong Lee	Bahman Teimourian
E.R. Brumskine-O'Tang	Patrick David Mansfield	Edward Stevens Urban
Johri Russell Burton	Sanford Max Markham	Julia W. Valdez
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Ma Concepcion H. Deluna	Bernard McGibbon	David Thos Walker
Julio M. Depena	David Painter Mohr	Robert Foster Ward
Cathleen O. Doane-Wilson	Soma Narasimha Reddy	Timothy John Whalen
Morris Zacharias Effron	William Herman Rogers	Boondharm Wongananda
John Glancy	Margarette Brown Rogler	Janet Peterson Woodyard
Victor Gong	Larry Arnold Sheingorn	Ravi C. Yalamanchili
Patricia H. Gulbrandsen		

Miscellaneous (continued)

Self-Directed CME Activities

Maryland physicians' campaign against family violence, module one: domestic violence, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Maryland physicians' campaign against family violence, module two: child maltreatment, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat 1 AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

Fluorescein angiography conference, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month; 8:00 a.m. – 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.

Sinai Hospital of Baltimore medical grand rounds, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.

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▼ The eleventh documented case of probable mosquito-transmitted malaria in the United States since 1986 was recently reported in a 53-year-old migrant worker residing in Tift County, Georgia (*MMWR* 1997;46(12):264-266). Although additional routes of infection could not be conclusively ruled out, based on consideration of the factors of this case, it was determined that the person probably acquired infection in southwestern Georgia through the bite of a locally infected *Anopheles* sp. mosquito. The study concluded that factors contributing to the increased frequency of locally acquired malaria include increased travel by U.S. residents to malaria endemic areas and shifting U.S. immigration patterns. Physicians should consider malaria in all persons with unexplained fever, regardless of travel history and particularly during summer months.

▼ Although celiac disease is usually found in young patients with clinically evident malabsorption, a recent study suggests that celiac disease should be considered in patients with unexplained metabolic bone disease or hypocalcemia, despite the age of the patient (*Arch Intern Med* 1997;157:1013-1016). The study authors found that gastrointestinal symptoms may be mild or nonexistent in this population. The authors present a case series of fifteen patients who were examined for hypocalcemia, skeletal disease, or both, who were subsequently diagnosed with celiac disease. The mean age of the patients was 62 years.

▼ According to an article in the *Archives of Pediatrics & Adolescent Medicine*, junior high school students are taking over-the-counter pain medications without the knowledge of their parents (1997;151:449-455). Acetaminophen was the most commonly used medication among this group, and the most common reasons for taking the medications included headache; stomach pain; ear and throat pain; muscle, joint, and back pain; and menstrual pain.

▼ *Let sleeping dogs lie.* A New Hampshire woman contracted human rabies after being bit by a stray dog while visiting the country of Nepal; she died from "an illness characterized by rapid neurologic deterioration" (*MMWR* 1997;46(12):267-270). Although she washed the wound with peroxide and alcohol and presented for medical attention at a hospital in Sydney, Australia as advised, a rabies vaccine was not immediately available and she did not return the following day as instructed. She did not receive postexposure prophylaxis (PEP). On the first visit to a New Hampshire emergency room, the patient was prescribed anti-inflammatory and analgesic drugs and discharged. On second presentation, two days later, she complained of progressive difficulty breathing, throat spasms, nausea, and vomiting and reported severe pharyngeal spasms when she drank fluids or showered. Over the next twelve hours she developed increasing agitation, anisocoria, salivation, and worsening facial and pharyngeal spasms. She died six days later. (Also see *Md Med J* 1996;45(9):765-769.)

▼ Ninety-nine patients and their physicians were surveyed after discharge from an academic medical center with the diagnosis of acute myocardial infarction or pneumonia, regarding the patients' understanding of discharge information (*Arch Intern Med* 1997;157:1026-1030). Physicians indicated that they thought 95% of the patients understood when they could resume normal activities, yet only 58% reported this to be true. Similar responses were noted regarding a patient's understanding of potential side effects from medications. The study authors concluded that improvement is necessary in patient-physician communication regarding postdischarge treatment plans.



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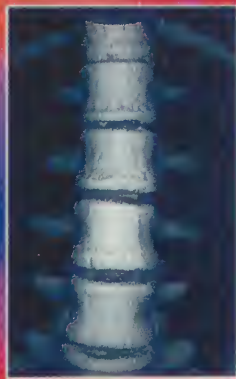
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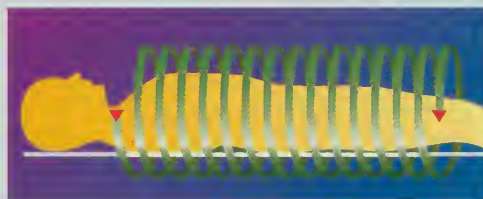
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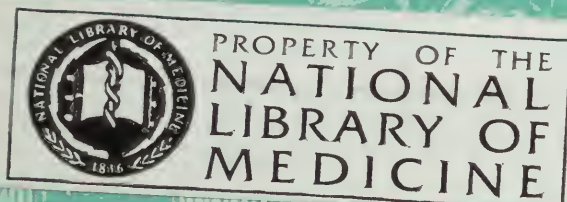
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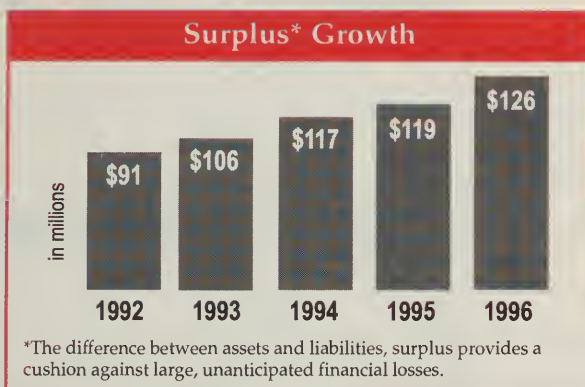
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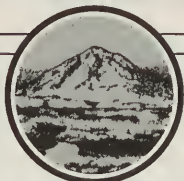
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Thursday	6	7	8	9	10	11	12	1	2	3	4	5	6	7	8	9
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The May/June 1997 issue is an outstanding one in that it focuses on malignant diseases of the skin. Every physician, no matter what field of practice, sees some of a patient's skin at each practice or social contact and,

therefore, we and the patients can benefit from this important information.

FRANK DAMAZO, M.D., P.A.

Dr. Damazo is a general surgeon practicing in Frederick, Maryland. ■

LETTERS TO THE EDITOR



The editorial board of the *Maryland Medical Journal* welcomes comments, criticisms, recommendations, and observations from all its readers. Please submit letters to: Editor, *Maryland Medical Journal*, 1211 Cathedral Street, Baltimore, MD 21201-5585

FROM THE EDITOR'S DESK

What's coming up?

September is "Women in Medicine" month. The *Maryland Medical Journal* will devote its September issue to articles by many female physicians in the Maryland area who are actively involved in medical affairs and who have attained leadership stature in their fields. They will discuss topics of current interest to physicians, including recent developments in anesthesiology, managed care and physician ownership issues, universal health insurance, and ophthalmology, among others. Additionally, we will present a review of the lives of two unusual female physicians – Helen Brooke Taussig and Celeste Woodward – whose medical activities spanned a roughly 50-year period during the middle of the twentieth century.

ERRATUM

In the July issue of the *Maryland Medical Journal*, the disclaimer statement was mistakenly omitted from the "From the BPQA" column on page 318. The following statement should have accompanied that article: *The opinions expressed in this column are those of Dr. Winchell and are not endorsed by the BPQA.*

The statements included in the article regarding frequency of discipline of International Medical Graduates were derived from data collected independently by Dr. Winchell. The BPQA has not verified the accuracy of her statements nor has the Board conducted or authorized analysis of disciplinary actions to determine whether or not the statements made are valid.

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Speak Out

The erosion of medical privacy today

Much ado about nothing? If anyone can possibly doubt the horror of the medical privacy crisis, let me quote from an Internet entry dated November 24, 1996, by Elizabeth Hatcher, M.D., Ph.D.

"I am a staff psychiatrist at the Menninger Clinic and a candidate in the Topeka Institute for Psychoanalysis. The extent to which third party payors demand violation of confidentiality was recently brought home to me when I learned from a hospital inpatient and her father that they had in the past been asked to send material from the patient's journal to the insurance company to document the fact that the patient was suicidal. Obviously, such a demand makes a mockery of such a patient's right to privacy in any basic human—not merely therapeutic—sense . . .

Does the need to document illness justify displaying a patient's private thoughts before strangers? . . . How is it . . . for patients to know that records about their worst nightmares are out there somewhere, in some company's [or state's] databank?"

Or what about this April 1997 Internet entry from Dr. Ivan Goldberg of New York City.

"I was once sent by mistake a set of reports that a managed care company sends to corporate employers. Not only were patients listed by name, but all the drugs that had been prescribed for them were listed."

No one can dispute the immeasurable positive contribution computer technology has made to health care, but it also poses a potentially insurmountable threat to medical privacy. How many computers now hold my patients' private and even stigmatizing diagnoses? And there will always be rogue employees willing to sell this information to nefarious information purchasers. How many people, authorized or not, have access to personal medical information?

We live in the Information Age, and the loss of personal privacy associated with legion databases may become THE civil rights issue of the next decade. Each year a higher percentage—now over two-thirds and growing—of medical and psychiatric care is "managed." And most managed care organizations (MCOs) share a boundless and reckless sense of entitlement to procure clinical information in the name of determining their own idiosyncratic and self-serving definitions of "medical necessity" and finding loopholes by which to deny care and increase profits. In fact, the codes of ethics of almost

Speak Out

all national health and mental health associations are being violated by non-consensual and patient-specific medical data bases and by MCO utilization review inquiries.

In December 1996, the American Psychiatric Association published a resource document titled "Preserving Patient Confidentiality in the Era of Information Technology." Here are a few statements from it:

"New information technologies should not be employed to stretch the limits of appropriate access that have been established in professional custom and law.

....Researchers should not maintain identifying information in computerized or electronic forms without patient consent.

....Security systems have been notoriously ineffective in preventing inappropriate access from those outside computer data systems.

....Professional organizations should continue to enforce vigorously ethical principles that require the confidentiality of patients' records to be maintained."

Privacy allows us to be who we are. The universally recognized and virtually common-law standard in this country for relinquishing confidentiality without consent has been compelling dangerousness, for example, child abuse, threats of suicide or homicide, or severe and imperiling contagion. No such situation exists with these economic-research-goal databases. No such situation exists with MCO utilization reviews. (Whereas MCOs technically obtain pro forma, blanket consents, these are meaningless, since few patients are aware of the scope of the disclosures, and consent is obtained under the duress of withholding treatment reimbursement if patients refuse to sign.)

Yet, MCO inquiries run rampant and databases proliferate at an alarming rate. For example, Maryland leads the nation with its universal non-consensual government-run (Health Services Cost Review Commission and the Health Care Access and Cost Commission) patient-specific databases, which contain ample individualized demographic information to render guaranteed confidentiality impossible. Soon we may have a nationwide "virtual" medical database as a consequence of the Administration Simplification aspect of the Kennedy-Kasselbaum 1996 Health Insurance Portability and Accountability Act.

Once a database exists, it is a monumental struggle to reverse the damage, as Marylanders have discovered: Charged politics and the whetted appetite of datamongers have defeated privacy advocates and health care professionals for the past two years in the Maryland General Assembly, in spite of an overwhelming show of support from Maryland's voting constituency for insisting upon patient

Speak Out

consent before database inclusion. Prevention of privacy intrusions is the most efficient approach, not correction, yet we are already in arrears.

Confidentiality is the very foundation of the doctor-patient relationship. The treatment relationship—the therapeutic alliance—simply cannot exist without it. Even the 1996 Jaffee vs. Redmond Supreme Court opinion on federal evidence admissibility acknowledged this.

However, the ethics of health care professionals are waning without our notice, eroded by the current Information Age demands. By routinely relinquishing personal patient information upon third party payor demand, we are in grave danger of the blindness and numbness of the defeated.

The younger the health care professional, the more these disclosures are an accepted part of health practice, not an ethical or moral concern. Young professionals have been taught managed care is a financial necessity to “save health care dollars”—never mind that managed care managers and owners are eating up those “saved” dollars in administrative costs, profits, and seven-figure chief executive officer salaries, as patients are getting less treatment than ever.

None of us can afford to be apathetic and worn down, to sink into a quagmire of feeling the loss of confidentiality is unimportant or beyond our control. We cannot afford to have a new generation of health professionals who do not understand the loss of confidentiality damages the treatment alliance and trust because they have not experienced the contrast between providing truly confidential care and care with permeable information boundaries. They will not know what has been lost.

Patients still believe their medical and psychological communications will remain confidential. They are not likely to disclose vital details freely unless they are certain no one not directly involved in their care will learn of them. Surveys estimate more than 25% of people would avoid seeking treatment altogether if it were not confidential. This may increase MCO profits, but it also disproportionately increases morbidity, disability, mortality, and overall societal cost by both fiscal and humanistic measures. Without full confidentiality, our consultation room becomes analogous to a personnel office job interview, with that interviewee’s wariness and limited disclosure.

Information system developers and insurance companies do not represent patients’ interests, as Sheri Alpert elucidates in her 1993 Hastings Center Report, “Medical Records, Privacy and Health Care Reform.” We must recognize that the number of people many states consider to have a legitimate interest in a person’s health care information is so great, it can effectively eliminate all medical privacy. We must acknowledge businesses, researchers, and health administrators see privacy as an impediment to accessing the data which they would like to use for management and research. We must understand information system developers find privacy a time

Speak Out

consuming, inelegant, and expensive concern. Information is power and power is a valuable commodity. Privacy gets in the way of power.

Health professionals and legislators must take the lead in protecting patient privacy. Balancing the need for medical privacy against the demand for information is not a medical or technical question, but ultimately a political one. Because patients are not well served by information systems designers and consumers, the political process must protect their privacy interests. We cannot look to data collectors to protect our privacy, because that is an inherent conflict of interest, no matter how reassuring their promises may sound. Instead, we must look to government to adopt the appropriate role of regulating and protecting data collection, and not for it to compete with data collection. Alas, this latter competitive position is the trend.

A May 1996 Time-CNN survey corroborated a 1993 Harris-Equifax survey that the highest priority of the American public is privacy. Eighty-seven percent of people responded they wanted their permission asked before their personal information was included in any database. Medical diagnoses and treatments are highly personal. They can and have been used in truly destructive, discriminatory ways, and often without the patient's knowledge, since data leaks usually do not come to public attention. When third party payors collect medical information, there is no guarantee who will see it, and in fact, their databases may become the source for wider dispersion to other databanks.

This country urgently needs an organized campaign to demand the restoration of medical privacy. Such a campaign may soon be initiated by the National Coalition for Patients' Rights, with the hope that it will be actively embraced by all health care associations. In the meantime, there are measures we can and must undertake on behalf of our patients and our profession:

- 1) We must share this ethical dilemma with our patients, explaining the privacy risks of information disclosures versus the reimbursement jeopardy if we don't divulge. Our patients must share in these decisions. We must collaborate with them on utilization review reports and other information provided to third party payors. If we know of existing non-consensual databases, such as the Maryland Medical Data Base, we must notify our patients.
- 2) We must document very thoughtfully, sensitively, sparingly, and with awareness of how far afield our information may one day go. The rule should be to document only that which is necessary and certain, even if that may increase our own risks in the event of potential malpractice claims (for which we have been taught to "document everything").
- 3) We should always refuse requests to deliver a full chart to third parties unless court ordered; chart summaries should be adequate for any other need, and the content of these summaries should be shared with the patient.

Speak Out

- 4) We must pay close attention to the details of consent forms, crossing-out irrelevant or undesirable clauses as well as adding protective limiting phrases about dates and recipients.
- 5) We should not use computerized documentation methods without our patients' knowledge and consent, and of course, take extreme precautions to safeguard our own records, electronic or otherwise.
- 6) Most important, we must all (patients and professionals alike) protest to and lobby legislators, patient advocacy groups, our health professions' organizations, insurance carriers, and those employers who purchase such privacy-intrusive insurance policies. We must also protest to our state insurance commissioner, attorney general and governor. We must make noise.

All of us can join the National Coalition for Patient Rights (1-888-44PRIVACY), and we can also join the National Coalition for Mental Health Professionals and Consumers (1-888-SAY NO MC). We can volunteer time, energy, and money, all of which can be very frustrating, but certainly better than the lie-down-dead alternative.

One thing is certain: Once privacy is lost, we will never get it back. We are at a frightening civil liberties crossroads, not just a crisis in medical care, and the reason is this: Tyrannical and repressive governments worldwide have in common the absence of personal privacy. Remember, privacy gets in the way of power.

Fighting NOW for privacy is our single best chance to reverse these ominous trends and to prevent further incursions, and it is a good time to fight. There are some early signs that the pendulum may be on the verge of swinging back towards primacy of the quality of patient care and the vital, necessary guarantee of confidentiality. Every person's effort matters.

Without confidentiality, there is no doctor-patient relationship.

JENNIFER KATZE, M.D.

Dr. Katze is in the full-time private practice of psychiatry in Towson. She has taken an active role in the battle to have consent be a legislated right before Marylanders have their information encoded in the Maryland Medical Data Base, and she is on the Confidentiality Committee of the American Psychiatric Association. This paper was delivered at the President's Symposium of the American Psychiatric Association annual meeting on May 21, 1997. ■



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Donald L. Lindberg, M.D., is one of the authors of a study indicating both healthcare practitioners and patients are increasing their use of computers. The authors conclude that the computer age has resulted in consumers who are eager for electronic information and comfortable sending private health information via the Internet. The article was published in the June 17 issue of *JAMA*. Dr. Lindberg is from the National Library of Medicine, Bethesda.

Claudia Kawas, M.D., is the principal author of a study published in the June 1997 issue of *Neurology* adding more evidence to the theory that women who use estrogen appear to have less risk of developing Alzheimer's disease. The researchers found that "women . . . on estrogen replacement therapy (ERT) had a 54% reduction in their relative risk of developing Alzheimer's disease." Although Dr. Kawas admits this study can't rule out that another characteristic common to women who use estrogen may be causing the reduced risk, she feels this study shows the need to explore estrogen's benefits. Dr. Kawas is associate professor of neurology at Johns Hopkins University School of Medicine.

Susan L. Furth, M.D., Neil R. Prove, M.D., M.P.H., M.B.A., Alicia M. Neu, M.D., and Barbara A. Fivush, M.D., are among the authors of a recently published report indicating that treatment centers seeing a higher percentage of pediatric patients are more likely to use a less invasive method of dialysis for children with end-stage renal disease. Study authors examined data on 1256 pediatric patients who underwent either peritoneal dialysis or hemodialysis at a single treatment facility for most of 1990. The study was published in the June issue of *Archives of Pediatrics and Adolescent Medicine*. Drs. Furth, Prove, Neu, and Fivush are from the Johns Hopkins Children's Center, Baltimore.

Jeanne McCauley, M.D., M.P.H., and colleagues published a study in the May 6 issue of *JAMA* finding that women abused as children will likely have physical and psychological problems as adults. Authors indicated that "it made little difference whether the abuse was sexual or physical." The study found that women abused as children were more likely to suffer depression, anxiety, low self-esteem, and drug abuse, and were more likely to attempt suicide than women who had never been abused. Dr. McCauley is from the Johns Hopkins University School of Medicine, Baltimore.

According to a study in the March issue of *Archives of Ophthalmology*, authored by **Dante J. Pieramici, M.D.**, and colleagues, propranolol, used to slow the heart rate, decreases tremors, pulse rate, and blood pressure without noticeable side effects, and significantly improves hand steadiness of surgeons during simulated eye operations. Seventeen eye surgeons volunteered to take part in the study. Dr. Pieramici cautions that while they are studying ways to improve the work of surgeons by looking at hand steadiness, they are not by any means advocating drug use by surgeons to reduce tremors. Dr. Pieramici is chief resident at Wilmer Eye Institute, Baltimore, and a retinal surgeon.

John Lawrence, M.D., is lead author of a study reporting on a technique developed to deliver genes to all cells of the heart. Using a disabled version of a common virus, the adenovirus, as a carrier, Hopkins researchers infected nearly 100% of the muscle cells of a rabbit heart with a new gene. Although more animal studies are needed, the researchers feel that with modern surgical techniques similar conditions should be able to be recreated for the human heart. The paper was published in the *Proceedings of the National Academy of Sciences*. Dr. Lawrence is assistant professor of cardiology at the Johns Hopkins University School of Medicine.

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Small cell carcinoma of the prostate: A case report and review of the literature

Cengiz Aygun, M.D.

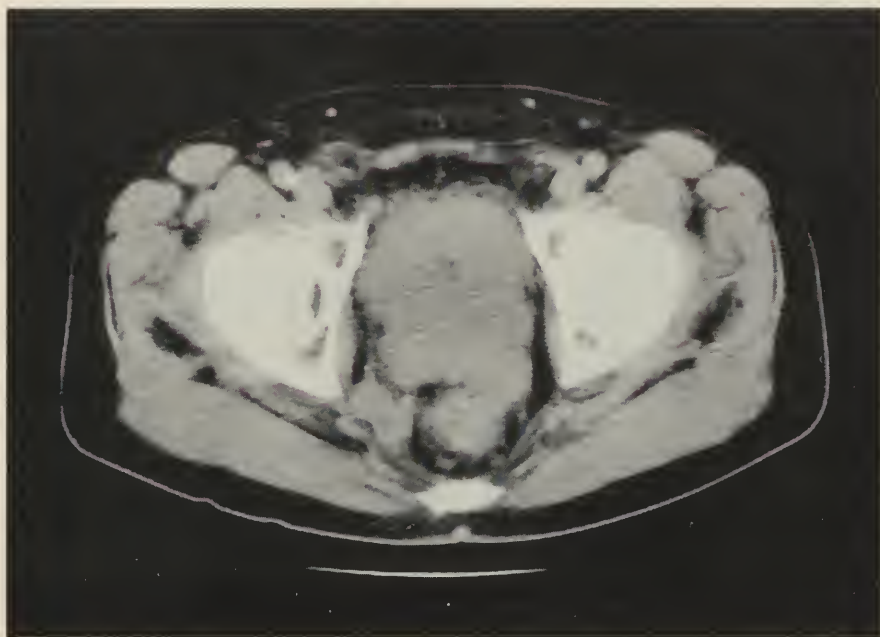
*Dr. Aygun is a radiation oncologist
with the Radiation Oncology
Affiliates of Maryland, Baltimore.*

ABSTRACT: *Primary small cell carcinoma of the prostate is rare. A case of primary small cell prostate cancer treated with radiation and chemotherapy is presented, and 33 previously published case reports are reviewed. Most of the patients (61%) had mixed tumors (small cell and adenocarcinoma) at diagnosis or had a history of adenocarcinoma of the prostate. Prostate specific antigen (PSA) data was available in 11 patients and was abnormal in 4 (36%). Once small cell carcinoma was diagnosed, 70% of patients had metastatic disease. Visceral metastases were common. Only one of seven patients responded to hormonal therapy, and two of eight patients responded to chemotherapy. Overall prognosis was poor.*

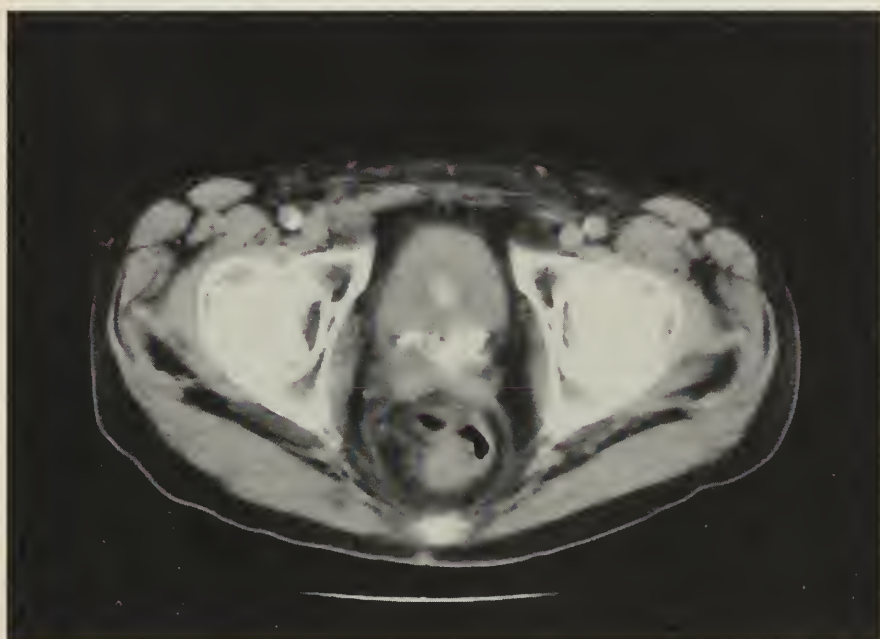
Extrapulmonary small cell carcinomas are unusual. Although these tumors can originate from any organ, prostatic origin is especially rare. The first case of small cell carcinoma of the prostate was published in 1977,¹ and only a handful of cases have been accumulated since then.²⁻²⁵ This report provides one addition to the literature on these rare cases of small cell carcinoma of the prostate and reviews the literature to date.

Case report

A 63-year-old man presented with progressive symptoms of prostatism. His medical history was unremarkable except for a heart murmur. There was a history of smoking over 20 years ago and he was a social drinker. The rectal exam was abnormal for an irregular, indurated prostate. A transrectal biopsy in June 1994 revealed a small cell carcinoma. He then underwent a transurethral resection of the prostate



▲ Figure 1a. Comparison of CT scans of the pelvis before and after (Figure 1b) treatment. ▼



(TURP) for urinary retention. The histopathology of the 25 grams of tissue which was removed revealed extensive involvement by an undifferentiated carcinoma characterized by relatively small cells with scanty cytoplasm and nuclear molding consistent with small cell carcinoma. In addition, there was a focus of moderately differentiated adenocarcinoma. Tumor cells reacted to immunohistochemical stain using neuron specific enolase and chromogranin. The metastatic work-up, including bone scan, computed tomography (CT) scan of the chest and abdomen, and bone marrow biopsy, was negative. PSA level was 2.4 ng/ml

(normal = ≤ 4). A repeat CT scan of the pelvis showed an enlarged prostate with extracapsular extension, pelvic adenopathy, and bilateral hydronephrosis (blood urea nitrogen [BUN]=92 and creatinine=10), a significant change with progression within seven weeks of the first CT scan (Figure 1a). Following a percutaneous nephrostomy, he received external beam radiation therapy and concomitant chemotherapy with Carboplatin and Etoposide. The radiation dose was 6420 cGy in 32 fractions to the whole pelvis, including a boost to the prostate gland from December 12, 1994, to February 2, 1995. Post treatment CT scan showed a complete response (Figure 1b). He then continued with additional chemotherapy. In November 1995, the patient developed brain metastases, but was free of disease elsewhere. He was treated with whole brain irradiation. A repeat TURP for urinary outlet obstruction in January 1996 was negative for malignancy. He then received monthly Lupron injections. In May 1996, he developed lung, liver, and bone metastases. PSA remained within normal limits. He died on June 12, 1996, 20 months after his diagnosis. An autopsy was not performed.

Review of the literature

There have been 33 case reports of prostatic small cell carcinoma published in the literature between 1980 and 1996.

The median age of patients was 65 (range 33 to 85). Of those with PSA data available, 64% (7/11) had normal values. Thirteen of 33 patients (Group I) had small cell carcinoma found in the prostate gland after a previous diagnosis of prostatic adenocarcinoma.

The small cell carcinoma diagnosis occurred at a median of 22 months following the discovery of the adenocarcinoma. All but one patient had metastatic disease present at the time of diagnosis of small cell prostate carcinoma; involved sites included bone (9), lymph node (9), liver (7), lung (6), central nervous system (CNS) (2), and pericardium (1). The prognosis of these patients was poor, with a median

survival of one month following the diagnosis of small cell carcinoma.

Seven of 33 patients (Group II) were found to have a mixed small cell and adenocarcinoma of the prostate gland at diagnosis. The adenocarcinoma component was poorly differentiated (4), moderately differentiated (2), and unknown (1). Thirteen of 33 patients (Group III) had pure small cell carcinoma of the prostate at diagnosis. Of the 7 patients with mixed small cell and adenocarcinoma (Group II) and 13 patients with pure small cell carcinoma (Group III), only 9 (45%) had localized disease at presentation. There were four patients with an elevated PSA, and three had mixed histology tumors. All patients with localized disease developed distant metastases during their disease course and died within 24 months of diagnosis (median survival = 5 months), (Table I). Metastatic sites included: bone (8), liver (10), lung (6), CNS (3), lymph nodes (12), and pericardium (2).

The 33 patients in Groups I, II, and III were treated with various modalities including hormonal therapy, chemotherapy, and combination therapies. Response rates to hormonal therapy were poor (one of seven responded). Only two of eight patients receiving chemotherapy had a response. Paraneoplastic syndromes, including SIADH, Cushing's, and Eaton-Lambert, were reported in 3 of the 33 patients (9%).

Discussion

It is estimated that small cell carcinoma of extrapulmonary origin comprises 0.1% to 0.4% of all other common forms of cancer.²⁶ Although neuroendocrine differentiation is frequently seen in prostatic carcinomas when immunohistochemical analysis is used, small cell carcinomas remain rare, comprising only 1% to 2% of all prostatic cancers.²⁷

There are three theories of histogenesis of small cell carcinoma of the prostate. According to one theory, small cell carcinomas of the prostate originate within the amine precursor uptake decarboxylation (APUD) system of Pearse. Initially, these cells were thought to be migratory from neural crest origin, but at present, it is suggested that these cells are of local endodermal origin.²⁷ Another theory proposes that small cell carcinomas are a product of dedifferentiation of an adenocarcinoma, and thus, part of a spectrum of prostate cancer rather than a separate biological entity.²⁸ The most widely accepted view is that small cell carcinoma originates from totipotential stem cells which have the ability to differentiate into both epithelial and neuroendocrine type carcinomas. Frequent coexisting glandular dedifferentiation with neuroendocrine cell neoplasia support this latter theory. Our case had foci of adenocarcinoma with small cell carcinoma.

Among the 33 case reports reviewed, 61% had both small cell and adenocarcinoma during their disease course.

Table 1. Clinicopathologic characteristics of 33 cases published from 1980 to 1996.

33 Patients with Small Cell Carcinoma of the Prostate

Age at Diagnosis: 33-85 (Median = 65)

◆ Group I (N = 13)

Prostate adenocarcinoma
(Previously Diagnosed)

subsequently
developed



Prostate small cell carcinoma
12/13 had metastatic disease
at diagnosis

Median Survival = 1 month

◆ Group II (N = 7)

Mixed Adenocarcinoma and
Small Cell Prostate Carcinoma

at initial
diagnosis



9/20 had localized disease
all developed distant metastases
within two years

◆ Group III (N = 13)

Pure Small Cell Prostate Carcinoma

Median Survival = 5 months

Given the rarity of prostatic small cell carcinoma, clear treatment guidelines are not possible. Chemotherapy is used sporadically and not uniformly, making its efficacy difficult to quantify. The response and overall survival rates appear to be lower compared to small cell carcinoma of the lung.

Conclusions

- ▶ Primary small cell carcinoma of the prostate gland is rare.
- ▶ Small cell carcinoma of the prostate gland has a tendency for rapid dissemination to visceral organs.
- ▶ PSA is not a reliable tumor marker except in patients with mixed tumors.
- ▶ Hormonal therapy has no role in the management of small cell carcinoma of the prostate.
- ▶ Because of the systemic nature of the disease, chemotherapy appears to be the mainstay of treatment for localized disease, with consideration for consolidative radiation therapy to the pelvis.

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Neuroleptic malignant syndrome and acute myocardial infarction: Case report and review

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ABSTRACT: *The neuroleptic malignant syndrome (NMS) is a potentially life threatening reaction usually observed following administration of dopaminergic antagonists (neuroleptic medications, e.g., phenothiazines, thioxanthenes, and haloperidol). NMS is characterized by mental status changes, muscle rigidity (and movement disorders such as dyskinesias and akathisias), leukocytosis, hyperthermia, and autonomic dysfunction. Because of the variants of this disease, the physician must remain alert to its possibility when confronted with emergency care of patients having received neuroleptics, particularly when hypermetabolic states are etiologically consequential in the development of other disease processes, for example, acute myocardial infarction.*

Case report

A 59-year-old mentally retarded gentleman was taken to the Sinai Hospital of Baltimore emergency department after his care-giver noted a one-day history of increasing rigidity, lethargy, listlessness, and non-communicative behavior. Previous medical history was remarkable for a stroke (with resulting right sided hemiparesis), an episode of diabetes insipidus, chronic schizophrenia, seizure disorder, and recurrent urinary tract infections. There was no surgical history, and the review of systems was non-contributory, except as previously noted. Family and social histories could not be obtained at time of admission. An allergy to Mellaril was noted. Medications upon admission included carbamazepine (800 mg BID), lithium carbonate (Eskalith^{Reg.}) (450 BID), amantadine (100 mg/bid), oxybutynin chloride (Ditropan^{Reg.}) (20mg TID) and the neuroleptic agent, thiothixene (Navane^{Reg.}) (10 mg/d).

Physical examination in the emergency room revealed a well-developed, well-nourished male appearing his stated age, who attempted to pronounce his name with severe difficulty. The temperature was 39.5 C, the pulse was 98, and the respirations were 20. The blood pressure was 158/104 mm Hg. His pupils were dilated and equally reactive to light and accommodation. No scleral icterus was noted. Mucous membranes were dry. Neck was supple and without jugular venous distention or hepato-jugular reflux. Lungs were clear to auscultation, and cardiac examination revealing a non-displaced point of maximal impulse (PMI) with regular rate and rhythm, without murmurs, rubs, or gallops. The abdomen was non-distended, bowel sounds were positive, and palpation revealed it to be soft and nontender. No cyanosis, clubbing, or edema were noted in the extremities. On neurologic examination there was rigidity in all extremities, with akathisia in the arms. He had a left Babinski and increased muscle tone bilaterally, with some cogwheeling in the upper extremities.

Laboratory data on admission. Computerized tomography of the head and chest roentgenograph were without acute findings. The lithium serum level was 1.5 mg/L (therapeutic, 0.5 to 1 mg/L); white cell count, 14 600/mm³; hematocrit, 38.9%; and platelet count, 275 000/mm³. The carbamazepine level was 11.3 mg/L (therapeutic, 4 to 12 mg/L). The Creatinine Phosphokinase (CPK-total) was 1718 IU (normal <235 IU), with CPK-MB, 72 IU (normal <5% of CPK-total). Electrocardiogram (ECG) revealed sinus tachycardia with normal intervals, left axis deviation, and non-specific ST and T wave changes. Electroencephalograph (EEG) was without obvious seizure focus. Urinalysis was within normal limits.

The patient was admitted to the intensive care unit to further assess a toxic encephalopathy, the differential diagnosis of which included infectious origin, anticholinergic delirium, NMS, lithium toxicity, and repetitive non-convulsive seizures.

On day two, the patient's temperature was 40 C, the pulse was 105, and the respirations were 40. The blood pressure was 150/90 mm Hg. Physical examination was remarkable for continuing rigidity, hyperthermia, and lack of communicativeness. Laboratory evaluation revealed CPK-total of 1861 IU, CPK-MB of 128 IU (7% of CPK-total). ECG displayed ST elevation in II, III, aVF, V₆, with acute ST depression in V₂₋₃, and relatively prominent R in V₂. A lumbar puncture was done and was traumatic. The results were glucose, 80 mg/L; protein, 54 mg/L; white cell count, 4; and RBCs, 1100. Cryptococcal, H. Influenza, N. Meningitidis, S. Pneumoniae, Grp. B Streptococcal Antigens, and

Gram stains were negative. Cardiac ECHO revealed an ejection fraction of 30%, with diffuse inferior and posterior hypokinesis.

Because of continuing rigidity, hyperthermia, elevated CPK-total, altered mental status, and leukocytosis, a diagnosis of NMS was entertained and the patient started on Bromocriptine, 2.5mg q8h. In light of the elevated CPK-MB, abnormal cardiac ECHO, and abnormally evolving ECG, the patient was also started on aspirin, a beta blocker, and an angiotensin converting enzyme (ACE) inhibitor for presumptive diagnosis of myocardial infarction. All antipsychotic/neuroleptic medications were discontinued.

Hospital course. After several days of bromocriptine therapy, the patient started to slowly respond to his name, and was noted to sleep most of the night. Rigidity and fever also improved. Thought process appeared coherent, but slow. The patient responded to questioning without offering elaboration. He denied depression and was not noted to be overtly psychotic; however, he continued to have impaired insight and judgment. Bromocriptine was stopped on day 10 of therapy.

Persantine Thallium stress test on day 23 showed a large area of myocardial scarring involving inferior, infero-lateral, and infero-apical wall with minimal residual peri-infarct ischemia in the infero-lateral myocardial wall. The patient continued to improve and was discharged 24 days after admission. Discharge medication included carbamazepine and lithium carbonate. Phenothiazines were withheld secondary to the patients diagnosis of NMS.

Discussion

NMS has been reported to occur in up to 1.0% to 3.0% of patients receiving dopamine antagonist therapy.¹ NMS may also be seen following acute or preexisting cranial injury.^{2,3} Interestingly, it has been observed both without muscle rigidity⁴ and/or hyperthermia, and appears to be age independent, having been observed in both the very young (e.g., previously healthy 6-year-old⁵) and elderly.⁶ Risk factors implicated include prolonged and/or parenteral administration of neuroleptics.⁷ Associated rhabdomyolysis may lead to hyperkalemia, disseminated intravascular coagulopathy (DIC), and acute renal failure.⁸ The reported mortality rate of "uncomplicated" NMS is as high as 38%, with death usually resulting from cardiovascular collapse.⁹ Development of myoglobinuria and/or renal failure are predictive of a higher mortality rate, approaching 50%.¹⁰

Recurrent NMS has been reported most commonly in patients with mental handicaps, particularly mental retardation.^{11,12} Perhaps this reflects a continuing need for neuroleptics in this relatively underserved population.

Because of elevated (total body) oxygen demands during the hypermetabolic (i.e., hyper-sympathetic^{13,14}) state accompanying NMS, it is attractive to speculate that the increased cardiac output required further exacerbates myocardial oxygen demand, which in the setting of pre-existent coronary artery disease, may lead to resultant myocardial ischemia. We are, however, aware of only one previous report of acute myocardial infarction being associated with development of the NMS.¹⁵

Comparable pathogenic mechanisms have been purported to occur during chronic cocaine use. Cocaine may alter availability of dopamine either through transmitter depletion or decrease in the number of dopamine receptors.¹⁶ Rhabdomyolysis and hyperthermia following cocaine use also appear to be pathophysiologically similar to that seen during NMS. An analogous syndrome has been observed following the use of "ecstasy," a new "recreational" drug (3,4-methylenedioxy-methamphetamine [MDMA]).¹⁷

Treatment of the NMS includes prompt discontinuation of neuroleptic agent(s), aggressive hydration, dantrolene, or bromocriptine. In the case of severe hyperthermia, cooling blankets may become necessary to establish eutheria. A reevaluation of the need for neuroleptics should be made, and restarting these drugs done with extreme caution, if at all.¹⁸

Conclusion

The neuroleptic malignant syndrome is a relatively rare disease with pronounced morbidity and mortality. It has been demonstrated in patients receiving dopaminergic blocking agents as well as following acute cranial or multiple trauma. The case presented, although rare, should alert clinicians to consider the concurrent presence of NMS as an etiology of acute myocardial infarction in patients presenting with rigidity, hyperpyrexia, or hypothalamic dysfunction and a history of neuroleptic ingestion.

As the use of neuroleptics increases, a high index of suspicion is warranted, particularly in cardiac patients prescribed neuroleptics. Early diagnosis will allow for treatment of the hypermetabolic state, which places the patient with acute myocardial infarction at increased risk of cardiovascular collapse.

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ONCOLOGY... TODAY

Progress in the treatment of childhood cancer

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The success achieved in the last thirty years in the treatment of children with cancer is due to the coordinated efforts of basic scientists, clinicians, and allied health personnel along with the tremendous courage of patients and their families. A wide variety of advances have radically altered the prognosis for this group of patients. In this brief review, we will focus on a few areas that have been fundamental in the improved survival and quality of life in these children.

During the period of 1974 to 1993, there was a 26% reduction in the mortality rate of childhood cancer, whereas the incidence rate increased by 4%. It is estimated that approximately 8000 children per year in this country under age 15 will develop cancer,

and that by the year 2000, the number of childhood cancer survivors in this country will be 180 000 to 220 000.¹ The five-year survival data in **Table 1** are extracted from the National Cancer Institute Surveillance, Epidemiology and End Results (SEER) Program data on several of the common childhood malignancies and the time period in which the children were diagnosed.^{2,3} Given the improved therapies now available, it is expected that over 80% of children diagnosed in the 1990s can expect a greater than five-year survival.¹

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A pediatric team approach, incorporating the skills of the local physicians, radiation oncologists, pediatric surgeons, pediatric medical oncologists, specialized nurse practitioners, other pediatric subspecialists, rehabilitation specialists, and social workers, is imperative for ensuring optimal therapy. Since cancer in children is fortunately relatively rare, the institution of large-scale national and international group clinical trials designed to answer specific therapeutic and biological questions has been essential in improving therapy and minimizing late effects.

A fundamental approach to treating childhood cancer is risk-based therapy. Children who are likely to respond poorly to standard therapy are identified and treated more aggressively in hope of improved survival. This more aggressive therapy may, however, pose a greater risk for short- and long-term morbidity or even mortality. Conversely, children at low risk of relapse are identified and treated with less intensive therapy with the hope of decreasing morbidity and improving quality of life. This risk assessment is no longer based solely on traditional fea-

Table 1. Childhood cancer: Five-year relative survival rates (%) in children ages 0-19

Site	Year of Diagnosis					
	1960-63	1974-76	1977-79	1980-82	1983-85	1986-93
All sites	28.0	58.4	63.9	66.4	69.7	73.4
Acute Lymphoblastic Leukemia	4.0	48.8	62.7	66.5	66.8	76.4
Wilms' Tumor	33.0	73.6	76.8	85.3	86.7	91.4
Brain and Nervous System	35.0	55.2	56.7	56.8	63.4	63.6
Neuroblastoma	25.0	51.7	53.2	53.0	53.3	63.0
Hodgkin's Disease	52.0	85.4	87.9	87.1	88.1	91.0

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The object of this new series, Oncology Today, is to provide concise review of current oncologic topics for the non-specialist in the field.

tures such as age and extent of disease, but also on biochemical, immunohistochemical, and cytogenetic features of fresh tumor samples. Because of the need for fresh (unfixed) tissue, it is imperative that the diagnostic procedure be performed at an institution prepared to process the sample for these studies. For example, improved cytogenetic techniques have demonstrated specific chromosomal abnormalities in the tumor cells themselves that have strong prognostic significance. Leukemia patients with translocations of DNA from chromosome 9 to chromosome 22 (the Philadelphia chromosome), or from chromosome 1 to 19, have been shown to have a substantially poorer prognosis with standard antileukemic therapy, and are therefore treated with more aggressive therapy including, in some cases, bone marrow transplantation while still in first remission.⁴ Similarly, children with neuroblastoma whose tumor contains amplification of the oncogene *N-myc* are at high risk for relapse with conventional therapy,⁵ and a current trial conducted by the Children's Cancer Group is examining whether intensive therapy, including autologous bone marrow transplantation, will improve the outcome for these patients.

Risk-based therapy has also led to the reduction of therapy in certain groups of children identified at diagnosis as highly likely to be cured. For example, the National Wilms' Tumor Study showed first that radiation therapy was not needed in the youngest children with this kidney cancer and further showed that, with better chemotherapy, it was not needed in older children with limited disease.⁶ Similarly, years ago, infants with neuroblastoma were often treated with radiation and chemotherapy. Now, in-

fants with neuroblastoma with specific molecular and cytogenetic findings in their tumor cells (e.g., lack of amplification of the *N-myc* oncogene) are merely observed with the high expectation of spontaneous regression. There are also tumor cell chromosomal abnormalities that predict for a good prognosis. Children with B-progenitor cell acute lymphoblastic leukemia, whose leukemic cells contain trisomy 4 and trisomy 10 (about 15% to 20% of patients), have a greater than 95% four-year event-free survival.⁷ As a result of these studies, patients in this group are currently treated with a less intensive regimen of antimetabolite therapy (6-mercaptopurine and methotrexate), which should result in few significant late side effects.

New developments in the fields of drug design and supportive care have also clearly had a positive impact on these patients. New chemotherapeutic drugs such as etoposide (VP-16), ifosfamide, and carboplatin have led to significant improvements in the outcomes of embryonal tumors such as Ewing's sarcoma, neuroblastoma, rhabdomyosarcoma, and germ cell tumors.⁸ Other newer agents, such as the topoisomerase I inhibitor, topotecan, seem to be active in brain tumors, sarcomas, and neuroectodermal tumors.⁹ Overcoming drug resistance, particularly multidrug resistance, remains a major challenge. Cells expressing the multidrug resistance phenotype often contain amplification of the gene encoding *mdr-1*, a drug efflux pump whose expression seems to correlate with the level of drug resistance.¹⁰ Methods for reversing these cellular changes have been sought, employing, among others, cyclosporin A or calcium-channel blockers like verapamil.¹⁰ Current

Pediatric Oncology Group phase II trials in children are examining whether cyclosporin A can reverse multidrug resistance. Since most anticancer drugs have a steep dose-response curve, the administration of anticancer drugs at maximal dose intensity (i.e., at the maximally tolerated dose and the shortest possible intervals) has gained wide acceptance¹¹ and has been the rationale for very-high-dose chemotherapy approaches with autologous bone marrow (BM) or peripheral blood stem cell (PBSC) rescue, particularly in solid tumors.

This type of intensive therapy requires a wide array of supportive measures. The introduction of hematopoietic growth factors, such as granulocyte- and granulocyte-monocyte colony-stimulating factors (G- and GM-CSF), have permitted the administration of higher doses of myelosuppressive therapy with a lower risk of profound and prolonged neutropenia, thus reducing the infectious complications seen in these patients.¹² It is anticipated that thrombopoietin, a growth factor involved in platelet production, will be clinically available in the near future to alleviate the need for recurrent platelet transfusions. The routine use of central venous catheters has substantially reduced child anxiety related to phlebotomy and other procedures as well as permitting the administration of total parenteral nutrition to appropriate individuals. The availability of improved antibiotics has clearly decreased the incidence of sepsis-related morbidity and mortality, but this success must also be tempered by the emergence of organisms resistant to multiple antibiotics, such as vancomycin-resistant enterococci.

These are but a few of the medical

and scientific advances that have improved survival and the quality of life for children with cancer. As our knowledge about the molecular biology of malignant transformation and tumor progression grows, new agents designed to interfere with these steps (biological response modifiers) are likely to gain more importance. This path from the laboratory bench to the bedside and back again, in the setting of randomized clinical trials, is essential for the continued development of new therapy.

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C. EVERETT KOOP, M.D., TO BE KEYNOTE SPEAKER AT BCMA CELEBRATION

The members of the Baltimore County Medical Association invite the medical community to help celebrate their centennial. C. Everett Koop, M.D., will be the keynote speaker at a dinner to be held at the Sheraton Baltimore North on Wednesday, September 10. Dr. Koop will speak on "The Doctor-Patient Relationship in the Era of Managed Care." Tickets are \$50 per person. Reservations are required. Please call the BCMA office at 410-296-1232 for further information.

When people with developmental disabilities present to community practitioners

by Lisa Hovermale, M.D.

Abstract

This article describes the problems involved in the global medical assessment of persons with developmental disabilities who present with behavioral changes. Solutions offered include anticipating the difficulties in treating this group and taking steps to insure adequate information is provided. Community providers are cautioned against assuming behavioral changes in this population are psychiatric in nature. Case examples illustrate the quality of life improvements that can result from relatively minor medical interventions in persons initially thought to have psychiatric difficulties.

Persons with developmental disabilities have a greater number and variety of health care needs as compared with those of the same age and sex in the general population."^{1,2} Establishing a diagnosis and finding the community resources to treat persons with developmental disabilities complicated by medical or psychiatric problems is extremely difficult. Yet, the solution to the presenting problem will almost always be medical, psychiatric, or behavioral. Finding that solution can vastly improve the life of the patient and those who care for him or her.

A young, nonverbal man with severe to profound mental retardation presents to the emergency room with the new, self-abusive behavior of repeatedly slapping his face on the left cheek area with great intensity. He is triaged to psychiatry because of his aberrant behavior. A visual exam of his mouth reveals obvious dental caries. An x-ray is obtained with great difficulty due to the patient's agitation. Multiple abscesses are seen. The behavior resolves completely after the abscessed teeth are pulled and the patient is treated with antibiotics. (The psychiatrist suffers vocal cord stress secondary to the "discussion" required to get this patient seen by persons who could diagnose and treat his problem.)

Confounds to finding the appropriately prepared provider

When young, the developmentally disabled are treated by pediatricians. Pediatricians tend to be well versed on the medical and behavioral issues associated with development disabilities and are adept at dealing with patients

This article is the third in a series that will explore various aspects of treating patients with developmental disabilities.

that, while often loud, communicate little except through behavior. They also tend to be terrific advocates for their developmentally disabled patients' rights to medical interventions. As the developmentally disabled move into adulthood, there is no specialty specifically trained to deal with their special needs or to advocate for those persons who cannot speak for themselves. Any distress, no matter what the source, can manifest as agitated or disruptive behavior in persons with major deficits in cognitive ability and communication skills. The term "diagnostic overshadowing"³ refers to the phenomenon of all such manifestations being attributed to the obvious trait of mental retardation rather than a state of psychiatric illness. Thus, when a person with developmental disabilities presents to a clinician or emergency room with an ill-defined complaint, with or without a disturbing or disruptive behavior, it can be very difficult to see past the mental retardation and/or behavior to look for another cause or exacerbating factor.

The presenting behavior can be quite disturbing to those in attendance; because the behavior is so obvious, psychiatry is thought to be the appropriate medical specialty. With the rising awareness of diagnostic overshadowing, some psychiatrists have become more willing to embrace this group of patients. Unfortunately, psychiatrists as a group are no more likely to have training in developmental disabilities than any other adult subspecialty physician. They must be motivated to acquire relevant continuing medical education to be effective. Those psychiatrists willing to work with this population must also resist another type of diagnostic overshadowing—assuming the problem is behavioral or psychiatric while overlooking the possibility the source of the difficulty is medical. This can occur when the treatment becomes focused on stopping the behavior rather than understanding it. The dangers in assuming all behavior in this group of patients is due to a psychiatric or behavioral sources rather than possible medical causes are manifold, but essentially, their suffering and the distress of their caretakers is prolonged and their life is potentially endangered. (Not to mention that it usually costs more to treat the wrong diagnosis.)

Confounds to adequate assessment

Attempts to understand developmentally delayed persons presenting with behavioral problems are often ham-

pered by a lack of information. Due to the developmental disability, the patient's verbal ability is often limited or nonexistent. The customary review of systems that clinicians use to focus an evaluation can be unobtainable. In patients who do have communicative ability, the information provided may be simply unreliable.⁴ If the person is still at home, then family can often provide an overview of changes in the patient's life or previous medical history. If the person is in a group home, obtaining this history can be much more problematic. Staff turnover in group homes can be rapid, and staff frequently remember only what they have witnessed themselves. Because staff rarely have any medical training, what they do report is open to wide interpretation. Sometimes, information offered by one staff member is contradicted by the next staff person who transports the patient. Records and charts are often voluminous, poorly organized, and incomplete, if available at all. Getting a succinct statement of what the problem is, when it began, and if it ever occurred before can take hours of unreimbursable effort and still produce a questionable product. In the end, clinicians willing to treat this population on the basis of limited information, a gestalt, or a theory must contend with the issues of whether this person can provide informed consent and if not, who will?

The dilemma of symptom nonspecificity is a particular problem among the developmentally disabled. Many developmentally disabled persons have behaviors or idiosyncrasies that are an ordinary part of their behavioral repertoire. These may include self-stimulation, stereotypy, self-abusive behaviors, or rituals. These behaviors exist without ready explanation and often occur at a baseline rate. In addition to the frequency of the behavior, the intensity must be considered (i.e., he is hitting himself the same number of times per hour but a lot harder each time). The only clue that the person is experiencing discomfort may be "baseline exaggeration."⁵ This concept has been used to better characterize psychiatric symptoms but can be just as useful to establish the medical diagnosis. It is important to remember there are many conditions in which the subjective experience of pain is the only symptom. It is incumbent upon the clinician to include these in the differential diagnosis of developmentally disabled persons presenting with a change in behavioral baseline. Clearly, one must know what the baseline is before it can be determined that there has been an exaggeration. It also must be established whether this

change in the baseline represents a short-term phenomenon or a long-term trend. Obtaining and synthesizing this kind of data requires expertise that most direct care workers do not possess. Although behavioral specialists and psychologists are often seen as only working with psychiatrists, the information they accumulate can prove invaluable to general medical clinicians as well.

Self-injurious behavior is a common response to the stress of being uncomfortable or in pain among the developmentally disabled. It has been suggested that self-injurious behavior may serve to modulate the pain or that the self-abuse may be the only means powerful enough to communicate the person's need for relief.⁶ One need only pause to consider the variety of ordinary pains (e.g., earache, headache, migraine, constipation, heartburn, backache, menstrual discomfort, gas, perimenopausal symptoms, ruptured ovarian cysts, ordinary anxiety) that are easily reported and remedied in a normal person's life. These same problems can prove overwhelming to a person totally dependent on someone else for many of their needs and whose pain may be communicated only through their behavior. If a medical basis is entertained as possible cause, the possibility is too often discarded if the person does not have a conspicuous response to the first round of treatments. In normal populations, conditions involving subjective reports of pain can often prove resistant to relief and treatment must progress to more complex strategies. In persons unable to complain effectively, or to locate pain well, this option can easily be lost.

In the current medical environment, where the average time spent with a patient is very brief, accumulating and understanding this data can be daunting to most community clinicians. The effort can be frustrating and unprofitable, at many levels, to the clinician. It must be conceded that a major factor limiting the availability of community resources is the economic disincentive. To treat this population effectively and efficiently, services must be provided in a setting prepared to deal with the associated logistical and legal problems. In a fee-for-service setting, the detailed assessment necessary would likely be provided at the expense of the personal time of the provider. These patients can be quite disruptive to the office routine due to behavioral responses to stress. It must be remembered that going to the doctor is stressful for everyone, but is much more so if you have limited capacity to understand why you are uncomfortable and anticipate what is going to happen next.

Possible solutions

One solution is for the clinician to establish an ongoing cooperative relationship with the care providers, so it can be clearly understood what information they must obtain before arriving at the clinician's office. Sovner has written on this topic as it relates to psychiatric diagnosis, and has provided templates useful in collecting the data.⁴ The same data needed to establish a psychiatric diagnosis may be helpful in establishing a medical diagnosis. He has suggested obtaining a biographical (and/or symptom) time line, a sleep chart, and a behavioral incident record. In addition, a menses record, a weight record, and some comment on bathroom habits can be useful in beginning an evaluation. Staff from all three shifts and from every setting in which the developmentally disabled person is cared for must understand the necessity and reason for this often tedious record keeping. Obviously, a large educational investment must be made to the direct care staff to obtain the necessary data.

First steps

Establishing a medication time line to correlate with behavioral records can be a critical first step. Current medications must be carefully scrutinized because drug side effects can result in behavioral deterioration. Anticonvulsants and psychotropic agents are the most commonly prescribed medications in this population, and both can produce side effects that result in behaviors inexplicable at first glance. Streamlining drug regimens to eliminate those that have not resulted in a discernible improvement is paramount. Finding more user-friendly options for those classes of drugs which cannot be discontinued becomes an ongoing goal. The following case illustrates the benefit that can result from this effort. It demonstrates the particularly unfavorable trade-off of seizure control for behavioral dyscontrol that phenobarbital can present.⁷

A woman with mental retardation has spent most of her life in an institution. In her late thirties, she is discharged to a group home in the community where she lives with eleven other disabled persons. Her discharge medications include phenobarbital and Dilantin for a seizure disorder. She has taken these medications as long as anyone can remember for seizures diagnosed in childhood. Her behavior quickly becomes problematic in the group home. There are

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A 69-year-old woman with hemoptysis, bilateral alveolar infiltrates, and microscopic hematuria

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PRESENTATION OF CASE

History. A 69-year-old woman presented to the emergency department with shortness of breath. Her symptoms began one week prior to admission and were associated with fatigue, malaise, and subjective fever. Two days prior to admission she developed a cough, with one episode of scant hemoptysis. She denied chest pain, purulent sputum production, prior constitutional symptoms, contact with persons with

known tuberculosis or other illness, or foreign travel. She kept no pets.

She had a medical history of atrial fibrillation, for which she was treated with digoxin and warfarin, and hypothyroidism, for which she was treated with levothyroxine. She had previously worked as a sales clerk in a retail store, but was retired. She smoked

one pack of cigarettes per day from age 21 to age 59, at which time she quit. She did not use alcohol.

Physical examination. The patient was afebrile, the pulse was 60, respirations were 16, and the blood pressure was 160/100 mm Hg. The conjunctiva were pale. No cervical or supraclavicular adenopathy was present. The rhythm of the heart was irregular, but no other abnormality was present. Inspiratory crackles were present in the mid portions of both lungs on auscultation. No expiratory wheezing was heard. The remainder of the examination was normal.

A sample of arterial blood, while the patient was breathing room air, yielded a pH of 7.38, a $p\text{CO}_2$ of 30, and a $p\text{O}_2$ of 47. The urine was cloudy and red, with an estimated protein content by dipstick of >300 mg/dl. On microscopic examination, the white and red blood cells were too numerous to count, and no casts or crystals were noted. The erythrocyte sedimentation rate was 65 mm/hr. The total serum protein was 6.3 gm/dl, with an albumin fraction which was low at 3.1 gm/dl. Serum transaminases, lactate dehydrogenase, and bilirubin levels were all normal. Other serum

Table 1. Hematologic and Chemistry Values

▶ Hematocrit	23.8%
▶ Mean Corpuscular Volume	91 fl
▶ White Cell Count	11,200/mm ³
▶ Neutrophils	78%
▶ Band forms	11%
▶ Lymphocytes	10%
▶ Monocytes	1%
▶ Platelet Count	293,000/mm ³
▶ Serum Sodium	133 mg/dl
▶ Serum Potassium	3.6 mg/dl
▶ Serum Chloride	100 mg/dl
▶ Serum Bicarbonate	18 mg/dl
▶ Blood Urea Nitrogen	57 mg/dl
▶ Serum Creatinine	5.7 mg/dl
▶ Serum Glucose	121 mg/dl

CASE RECORDS

chemistries and hematologic values are shown in **Table 1**.

A chest x-ray (**Figure 1**) showed bilateral alveolar opacities in a peri-hilar distribution with sparing of the apices and lower lobes. Further laboratory testing and a diagnostic procedure were performed.

DIFFERENTIAL DIAGNOSIS

Our patient is a 69-year-old woman who presented with a subacute illness characterized by pulmonary and renal dysfunction. Of note, she did not appear severely ill at presentation. Based on the appearance of her chest x-ray, I will assume that she deteriorated more rapidly following admission. This patient has a disorder which predominantly affects two systems—the lungs and kidneys. I presume that she has a history of long-standing hypothyroidism since she was treated with thyroid replacement therapy. I do not believe that the present illness is directly related to the thyroid disease. One should note that severe hypothyroidism has been associated with mild elevations in serum creatinine levels secondary to increased creatinine production;¹ however, the increase in serum creatinine concentration caused by hypothyroidism does not reach the levels seen in this patient, and hypothyroidism does not affect blood urea nitrogen (BUN) or produce hematuria. Therefore, I will

exclude the history of thyroid disease from my consideration of the pulmonary and renal disease.

The patient's pulmonary disease is characterized by dyspnea, hypoxemia, and diffuse alveolar infiltrates that have the appearance of coalescing indistinct nodules without cavitation. She had crackles on auscultation of the chest and possibly some hemoptysis, although this occurred while the patient's anticoagulation therapy was supertherapeutic. Reports of purulent sputum, wheezing, and extra cardiac sounds were notably absent. The arterial blood gas analysis revealed a widened alveolar-arterial oxygen gradient.

The terminology used to describe chest x-ray abnormalities can be quite misleading when used imprecisely. There are four patterns of pulmonary parenchymal involvement: alveolar, interstitial, vascular, and destructive. Destructive implies bullae, cavities, and focal scarring with the honeycomb pattern seen in the end stage. Vascular patterns refer to the normal vascular and lymphatic structures within the thorax, the bronchovascular bundles, the lymphatics, or the lymph nodes.

The remaining two terms, interstitial and alveolar, are most often misused by non-radiologists. These patterns can best be understood by considering the normal lung anatomy. The secondary lung lobule includes all airways distal to the lobular bronchus which includes several acini.

The acinus, or primary lung lobule, is the lung unit distal to the terminal bronchiole and includes respiratory bronchioles with alveolar outpouchings, alveolar ducts, and the terminal alveolar sacs. These acini are three to five mm in diameter and are separated from each other by very thin septae, which in normal lungs are below the resolution of the x-ray beam. In x-rays of normal lungs, each air-filled acinus appears black. The surrounding connective tissue is too thin to be seen on chest x-rays. Thus, the x-ray pattern of a normal lung is a black background with linear white structures rep-



Figure 1a & 1b. PA chest X-ray demonstrating peri-hilar opacities with sparing of the apices and bases.

representing vascular structures, bronchovascular bundles, pulmonary veins, lymph nodes and fissure planes. If the septae surrounding each acinus are thickened because of fluid, collagen, or inflammation, the x-ray can discriminate distinct air-filled black circles separated from each other by white lines. This produces a lacework pattern termed an interstitial pattern, also called a reticular or periacinar pattern. Interstitial patterns may blur slightly, but do not usually obliterate margins of adjacent structures. If the terminal airways themselves are filled with fluid, inflammatory cells, or cellular debris, or are collapsed, the chest x-ray will appear as a coalescence of white circles. Depending on the pathophysiologic process, these white densities can fill a lobe or an entire lung. They may be patchy or nodular, or appear as large coalescing nodules. In contrast with the interstitial pattern, alveolar processes cause the loss of borders of adjacent structures, the so-called silhouette sign. In the honeycomb lung, or the end-stage destroyed lung, there is thickened connective tissue around the air-filled lung units as seen in the interstitial pattern, but the lung units are much larger and often distorted, either by overdistension or destruction of septae between smaller lung units or by traction secondary to fibrosis.

Our patient's admission chest x-ray shows an alveolar pattern that appears to be a coalescence of many indistinct nodules. The borders of the mediastinal structures are lost and there is a central distribution of the infiltrates. I would classify these changes as an alveolar filling pattern. There is no sign of cavitation, pleural effusion, or pneumothorax.

The renal involvement in our patient is characterized by elevation in both creatinine and BUN, microscopic hematuria, and marked proteinuria, which I will assume is of recent onset. Even with an absence of detectable cellular casts in the urine, this constellation of findings suggests a destructive renal process, involving at least the glomeruli and perhaps other parts of the nephrons. The elevated blood pressure and the low sodium may be additional nonspecific signs of renal disease.

The patient has a low hematocrit. The normal lactate dehydrogenase suggests that the anemia is not caused by ongoing hemolysis.

If the renal disease were chronic, it could certainly cause this degree of anemia, but I am assuming the renal disease is new. There is nothing from the history to suggest gastrointestinal blood loss, but there is a persistent loss of erythrocytes in the urine. Furthermore, as we will discuss, the patient may have sequestered blood in the extravascular pulmonary parenchyma. One must also consider if the low hematocrit is part of a systemic disease which also involves the bone marrow; however, the normal platelet count makes this explanation less likely. The patient has some nonspecific findings of ongoing systemic inflammation, as evidenced by a mild leukocytosis, with an elevated percentage of band forms and a high erythrocyte sedimentation rate. Pertinent negative data include a lack of eosinophilia, which will exclude at least one possible diagnosis, and normal serum transaminase levels, which effectively excludes significant acute hepatocellular injury.

Diffuse alveolar hemorrhage usually presents with abrupt onset of dyspnea, hypoxemia, and an alveolar-filling pattern on chest x-ray. Hemoptysis may be mild or absent, since the source of bleeding is the distal airway and blood can be sequestered in the terminal air spaces rather than being cleared to the central airways and expectorated. If the source of the hemorrhage in this patient were the more proximal airways, the hemoptysis should have been more severe.

Alveolar hemorrhage is usually suggested by the clinical and roentgenographic picture. Further evidence of alveolar hemorrhage is provided by one or more of the following procedures. Open lung biopsy or transbronchial biopsy will show an accumulation of erythrocytes and (if the hemorrhage is ≥ 2 days old) hemosiderin-laden alveolar macrophages. Bronchoalveolar lavage will also show erythrocytes and hemosiderin-laden alveolar macrophages. Repeated measurement of pulmonary diffusion capacity (diffusing capacity for carbon monoxide or DLCO) can also provide clues to the presence of alveolar hemorrhage.² This test measures the absorption of carbon monoxide (CO) during a held breath. The large mass of extravascular blood in alveolar hemorrhage serves as a sink for

CO, increasing the measured DLCO. Because the blood is not circulating, its CO binding sites are saturated and will not be available for binding CO in subsequent DLCO maneuvers. A pattern of an elevated DLCO value, which progressively diminishes with repeated measurements, suggests the presence of extravascular pulmonary blood. Finally, the use of magnetic resonance imaging has been reported to aid in the diagnosis of alveolar hemorrhage, but this technique is not yet widely used for this diagnosis.

This case nicely demonstrates the importance of identifying the key constellation of signs, symptoms, and laboratory data on which to base the differential diagnosis. Failure to do so will result in either a differential diagnosis so broad as to be worthless or to omit the correct diagnosis. The differential diagnosis of oxygenation failure with the chest x-ray changes seen in our patient is very long. Therefore, we have to add some of the key features of this case to allow us to develop a more focused differential diagnosis. I believe the key elements of this case are its subacute nature, the diffuse pulmonary infiltrates and hypoxemia, the renal failure, microscopic hematuria and proteinuria, and the signs of systemic inflammation. In combining these factors, we can reduce the possible diagnoses to a handful of candidates.

I would like to first address and dismiss infectious etiologies. In the absence of sepsis or an adverse effect from antibiotic therapy, bacterial and viral pneumonias do not cause renal failure, with the exception of *Legionella pneumophila* infections.³ Infection with this pathogen also characteristically causes low plasma sodium levels,

usually have a productive cough. Thus, *Legionella pneumonitis* would be an unlikely explanation of our patient's presentation.

A paraneoplastic syndrome, including nephrotic syndrome, has been reported, albeit rarely, in bronchogenic adenocarcinoma⁴ and is believed to be caused by immune complexes containing tumor antigens. Thus, adenocarcinoma of the lung with widespread pulmonary metastasis could explain the patient's pulmonary and renal disease, but the acute onset of the respiratory symptoms does not favor this diagnosis. Bronchoalveolar cell carcinoma, which would be more consistent with the chest x-ray appearance, has not been identified with this paraneoplastic lesion as far as I know. Alternatively, renal cell carcinoma, with locally advanced disease causing renal failure, could metastasize to lung and spread via the airway route to cause a chest x-ray pattern similar to our patient's x-ray, but again this scenario is quite unlikely.

I believe this patient has an immunologic disorder causing her pulmonary and renal disease. I will discuss these possible diagnoses (Table 2) and see how they fit the available clinical and laboratory data.

Goodpasture first described a syndrome of diffuse alveolar hemorrhage and glomerulonephritis in 1919 in an 18-year-old man who died 6 weeks after a presumed bout of influenza.⁵ Today, we realize that the active factor is an autoantibody targeted against glomerular and alveolar basement membrane.⁶ Patients, usually men between 20 and 40 years old, present with acute or subacute onset of cough, dyspnea, and hemoptysis. The disease occurs with equal frequency in older men and women. Renal and pulmonary disease are discovered coincidentally in 60% to 80% of the cases, but most of the patients who present with alveolar hemorrhage are current cigarette smokers. Our patient was not actively smoking. Demonstrating circulating anti-glomerular basement membrane (GBM) antibodies in the correct clinical setting is diagnostic for this disease,⁷ but results may take several days to be reported. Renal biopsy demonstrating linear immunoglobulin deposits along the glomerular basement membrane can provide the diagnosis more rapidly. Treatment of Goodpasture's disease consists of corticosteroids, cytotoxic agents,

as was seen in our patient. *Legionella* can cause microscopic hematuria, proteinuria and renal failure; however, the patients in whom this is reported are usually very ill at presentation, unlike our patient. Moreover, patients with *Legionella* infections are rarely afebrile and

Table 2. Causes of pulmonary-renal syndromes

- ▶ Goodpasture's syndrome
- ▶ Wegener's granulomatosis
- ▶ Pulmonary capillaritis
- ▶ Polyarteritis nodosa
- ▶ Polyangiitis overlap syndrome
- ▶ Pauci-immune crescentic glomerulonephritis
- ▶ Allergic angiitis and granulomatosis (Churg-Strauss syndrome)
- ▶ Mixed cryoglobulinemia
- ▶ Henoch-Schonlein purpura
- ▶ Hypersensitivity vasculitis

and plasmapheresis. Two-year survival is only 50%, with most deaths caused by diffuse alveolar hemorrhage or infection.⁸ Our patient is a bit old for this diagnosis, but otherwise a good fit.

Idiopathic pulmonary hemosiderosis is a disease of children, with only 20% of cases occurring in adults. In fact, the oldest reported patient with this disease was 62 years of age,⁹ 7 years younger than our patient. The disease usually presents as a progressive respiratory failure, abnormal chest x-ray, and iron-deficiency anemia. Renal involvement is rare and, for some clinicians, would exclude this diagnosis. The etiology is unknown, but histopathologic exam shows alveolar wall necrosis, intra-alveolar blood, hemosiderin-laden macrophages, immune complex deposition, and interstitial fibrosis. Treatment consists of immunosuppressive therapy and blood transfusions. In children, mean survival is three to five years, but mortality is much lower in adults with this disease. Our patient's age and her renal involvement make this diagnosis very unlikely.

Polyarteritis nodosa is a multisystem disease characterized by a necrotizing vasculitis of the small and medium size arteries in multiple organs, including kidney, liver, nervous system, skin, and joints, but rarely in the lungs.¹⁰ In fact, those cases of polyarteritis nodosa with lung involvement are probably misdiagnosed cases of Churg-Strauss angiitis¹¹ or the so-called polyangiitis overlap syndrome.¹² Thus, I will dismiss this diagnosis in our patient, in whom lung involvement is a major feature.

Anaphylactoid purpura or Henoch-Schönlein purpura (HSP) is a form of leukocytoclastic immune complex vasculitis characterized by the triad of purpura, arthritis, and abdominal pain. It is predominantly seen in children, but can also occur in adults.¹³ Glomerulonephritis and renal failure can occur, but respiratory involvement is rare. Our adult patient had none of the usual presenting signs and symptoms of HSP. So, I will dismiss this diagnosis as well.

A family of vasculitides characterized by granuloma formation are the most common type of pulmonary vasculitis. Of these, Wegener's granulomatosis is the most common diagnosis. Originally described by Wegener in 1939 as a necrotizing granulomatous vasculitis of the upper and lower respiratory tract and glomerulonephritis,¹⁴ it is now

seen in variable clinical presentations,¹⁵ including limited Wegener's granulomatosis involving only the lower respiratory tract.¹⁶ Wegener's granulomatosis presents most commonly in middle age, with systemic symptoms and either upper or lower respiratory symptoms, including cough, dyspnea, and hemoptysis. The onset is variable, ranging from insidious to abrupt. Pulmonary involvement is characterized as infiltrates in 63% of cases, multiple nodules simulating metastatic disease in 31%, and with cavitation in only 8% to 10% of cases.^{17,18} Upper airway involvement is a feature in 80% of cases of Wegener's, but occurs in less than half of patients in whom Wegener's granulomatosis presents as alveolar hemorrhage,¹⁹ which I believe occurred in our patient. Renal failure is seen in 15% of patients initially presenting with Wegener's. Diagnosis is based on the clinical presentation and open lung biopsy showing parenchymal necrosis, granulomatous inflammation and inflammatory cell infiltration with neutrophils, lymphocytes, plasma cells, and eosinophils. Blood vessel obstruction and infarcts occur in one-third of cases with an accompanying capillaritis and alveolar hemorrhage.²⁰ Unlike Goodpasture's disease, the renal pathologic findings in Wegener's granulomatosis are not sufficiently specific to be diagnostic. Demonstration of anti-neutrophil cytoplasmic antibodies (ANCA) of the "C" or cytoplasmic type, which are targeted against protease III, further supports the diagnosis. "P" or perinuclear ANCA are targeted to anti-myeloperoxidase and other cell proteins and are more commonly seen in non-Wegener's vasculitides in which capillaritis is more prominent.²¹ The etiology of this class of vasculitides is unknown, but the ANCA itself may contribute to the disease activity by activating neutrophils.²² Wegener's granulomatosis represents one of the great successes in treating immunologic disorders. Before the onset of therapy, the disease was uniformly fatal with survival of only a few months. With present therapy, corticosteroids and cyclophosphamide, complete remission is achieved in 75% to 90% of patients.¹⁹

Churg-Strauss syndrome, also called allergic granulomatosis and angiitis, was described in 1951 in a series of 13 patients presenting

with severe asthma associated with fever and hypereosinophilia.¹¹ Granulomatous vasculitis can affect small arteries in many organs, including lungs, peripheral nerves, skin, and heart, but it uncommonly involves the kidneys.²³ Our patient's lack of eosinophilia and wheezing and the low incidence of renal involvement with this disease make this diagnosis unlikely.

Lymphomatoid granulomatosis was described in 1972 by Liebow and colleagues as a unique form of pulmonary angiitis characterized by an angiocentric and angiodestructive infiltration of the upper and lower respiratory tract, skin, cen-

tral nervous system, and uncommonly the kidneys.²⁴ Chest x-ray abnormalities were indistinguishable from those in patients with Wegener's granulomatosis. T cells predominate in the cellular infiltrates, and lymphomas either predate or subsequently develop in many of these patients.²⁵ Remission is induced in half of the patients with immunosuppressive therapy. Lymphoma develops in most of the other patients. Because this disease rarely affects the kidneys, I will not consider this diagnosis further in our patient.

Liebow also described a probable variant of sarcoidosis which he termed necrotizing sarcoid granulomatosis.²⁶ This syndrome is characterized by confluent granulomas, similar to those seen in sarcoidosis, associated with minimally necrotic granulomatous vasculitis of the pulmonary vessels, pulmonary nodules, or infiltrates, without hilar adenopathy. Patients with this syndrome usually have a benign course. Renal involvement, when it occurs, is usually characterized by an interstitial nephritis and renal tubular acidosis. Renal failure in sarcoidosis is usually caused by extensive nephrolithiasis caused by increased serum calcium levels. The calcium level in our patient was normal. Therefore, I am also discounting this diagnosis.

Acute lupus pneumonitis is an uncommon feature of systemic lupus erythematosus (SLE), which most frequently occurs in younger patients with the disease.²⁷ Its onset can be acute or subacute and can cause alveolar hemorrhage in 1% to 2% of cases.²⁸ Histopathologic evaluation shows the typical findings of alveolar hemorrhage with small vessel angiitis, and in half the cases, immune complex deposition can be demonstrated. Alveolar hemorrhage in SLE has a high mortality rate (50% to 75%), with death from respiratory failure, despite treatment with corticosteroids and cytotoxic agents.²⁸ SLE involves many organ systems, including the kidneys as seen in our patient, but it is uncommon for this disease to initially present as a pneumonitis.

Mixed cryoglobulinemia, a systemic vasculitis caused by the presence of cryoglobulins in the circulation, can involve both kidneys and lung, but it usually includes purpuric skin lesions, hepatitis, and arthritis, none of which were seen in our patient. Furthermore, the lung

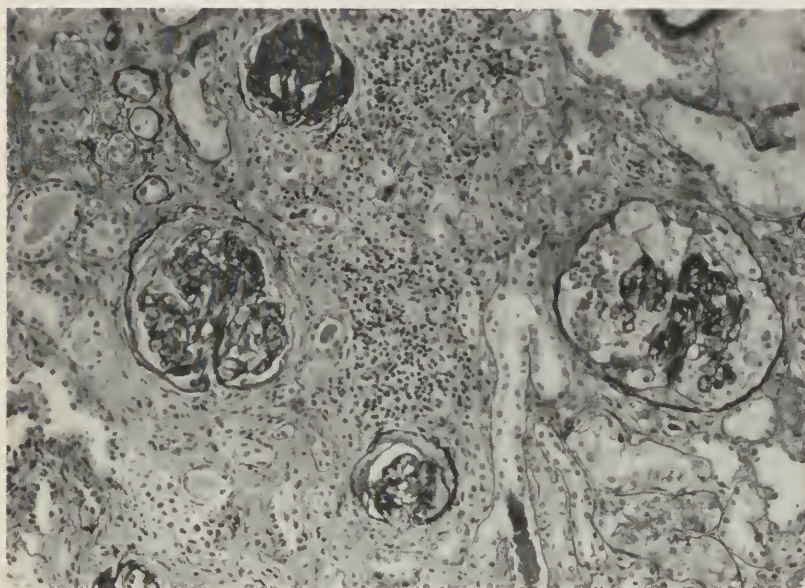


Figure 2. Light microscopy of kidney biopsy, hematoxylin, and eosin stain. The section shows four glomeruli in various stages of crescent formation, crowding of glomerular tufts, necrosis and sclerosis.

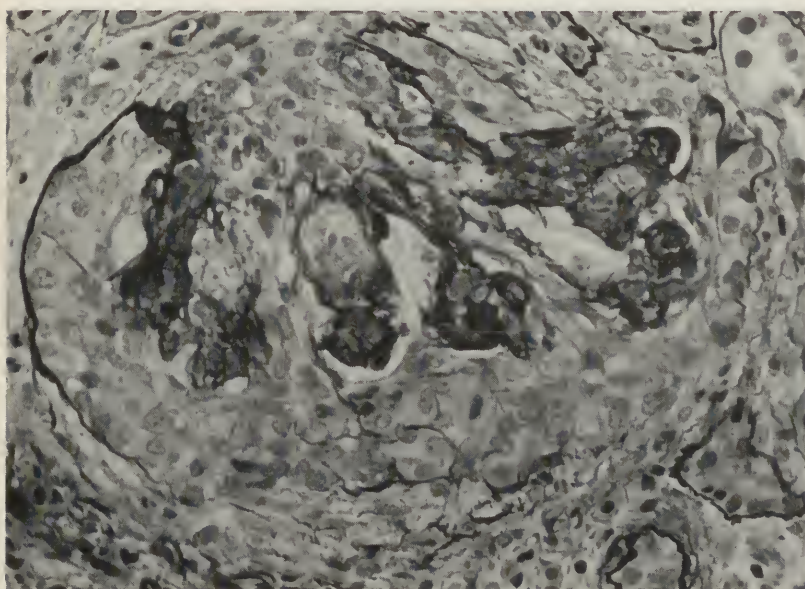


Figure 3. High power view of a glomerulus with well-developed epithelial crescent.

involvement in this disease is most often a chronic interstitial process,²⁹ rather than the subacute alveolar filling process which occurred in our patient. Sjogren's disease can present with both lung and renal involvement, but our patient did not have any symptoms that comprise the sicca complex, dry eyes and mouth. Furthermore, the lung involvement in Sjogren's syndrome is more chronic and interstitial in nature, as seen in mixed cryoglobulinemia. The mucocutaneous ulcerations in Behcet's disease can involve the larger airways and present with alveolar hemorrhage,³⁰ but our patient is quite old to experience an initial presentation of Behcet's, nor does she demonstrate any of the stigmata of the disease. Moreover, since the source of bleeding in Behcet's is from the more proximal airways, I would have expected more severe hemoptysis if the pulmonary disease was a manifestation of Behcet's disease. I think we can safely exclude these three diagnoses.

I have narrowed down the diagnostic possibilities for this patient's pulmonary-renal syndrome to three: Wegener's granulomatosis, systemic lupus erythematosus; and Goodpasture's disease. Although she lacked the evidence of upper airway involvement which is part of the classic triad of Wegener's, this can be absent in over 50% of cases of Wegener's granulomatosis. The subacute onset of disease can be seen in all three entities, but the age of onset and the nodular x-ray appearance of the pulmonary infiltrates best fit the diagnosis of Wegener's granulomatosis or a related vasculitis.

Clinical diagnosis. I believe the patient had Wegener's granulomatosis or a related pulmonary capillaritis. The positive laboratory test was probably an ANCA measurement, and the open lung biopsy likely showed the features of Wegener's granulomatosis or pulmonary capillaritis.

PATHOLOGIC DIAGNOSIS

A percutaneous biopsy of the right kidney was performed. Microscopic sections show marked pleomorphic glomerular changes. The various glomerular lesions are dominated and characterized by crescent formation in addition to changes in the glomerular tufts. Many glomeruli show fibro-epithelial crescents with compression of the glomerular tufts (**Figures 2 and 3**). Although most glomeruli are involved, there are occasional normal glomeruli seen (in a representative section, 20 of 35 glomeruli were globally or segmentally sclerotic). Other glomeruli show focal hypercellularity

and fibrinoid necrosis. Tubular changes are seen with vacuolization and tubular sloughing, and there are foci of tubulitis. There is marked interstitial fibrosis and interstitial inflammatory infiltrate by mononuclear and polymorphic nuclear leukocytes, as well as atrophy and tubular dilation. Immunofluorescence microscopy, performed at another laboratory, showed rare foci of C3 and IgG immunoreactivity within crescents and, more rarely, within glomerular segments. Peritubular linear C3 immunoreactivity was present. Fibrinogen immunoreactivity was present within the glomerular crescents and necrotizing foci, tubules, and interstitium. No significant IgA, IgM, or C1q immunoreactivity was noted. The light microscopic findings are consistent with a necrotizing glomerulonephritis with many crescents, and together with the immunofluorescence findings, this is consistent with a pauci-immune necrotizing glomerulonephritis.^{31,32,33}

Anti-basement membrane antibodies in serum were reported by the Corning Nichols Institute as being <5 units, which is considered normal. "C" type anti-neutrophilic antibodies were reported at <7 EU/ml, which is considered normal, but the "P" type ANCA was reported at 27 EU/ml (normal < 7). The pauci-immune pathologic appearance, the presence of ANCA antibodies, and the clinical course suggest either Wegener's granulomatosis or a microscopic polyarteritis. The presence of "P" type ANCA antibodies, and the absence of "C" type antibodies makes the diagnosis of microscopic polyarteritis more likely.

FOLLOW-UP

The patient was treated with cyclophosphamide and prednisone, which resulted in rapid and substantial improvement in her pulmonary infiltrates. Her renal function improved, although she continued to exhibit azotemia. Two months later, she developed severe pancytopenia, requiring cessation of cyclophosphamide and treatment with granulocyte colony stimulating factor. Three months after her initial diagnosis, she was admitted with recurrent pulmonary infiltrates, hypoxemia and fever, but refused repeat bronchoscopy, and was treated empirically for possible pneumonia, as well as recrudescence of her underlying illness.

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MARYLAND MEDICAL HISTORY

A long forgotten tribute to the “Emperor” recalled on his 200th birthday anniversary

Extracting from the enormous tapestry of medical history that thread which represents the development of surgery upon the thyroid gland allows one to view a slender, luminescent filament drawn from that portion of the entire panorama which is our surgical heritage. We learn that attempts to remove tumorous growths from the neck were recorded in European history as early as the Greco-Roman era and in the Middle East as early as the third century A.D.

Although much has been written about the operative treatment of thyroid growths and the accomplishments throughout recorded history of both greater- and lesser-known surgeons, there is no more carefully documented or scholarly account of all reported cases than that of William S. Halsted, M.D., published in 1920.¹

He declared, “The extirpation of the thyroid gland typifies, perhaps better than any operation, the supreme triumph of the surgeon’s art.” In that publication, it was clearly his intent not only to tabulate every published report from the world literature of surgical extirpation of thyroid goiter, but to record mortality and morbidity, bring attention to the pioneer resectionists by nation of origin, and cite those published cases which seemed to have escaped the scrutiny of prior reviewers. In doing so, he illuminated many long-eclipsed accounts of brave individuals who, with little or no experience to guide them, undertook to relieve the pain, airway encroachment, disruption

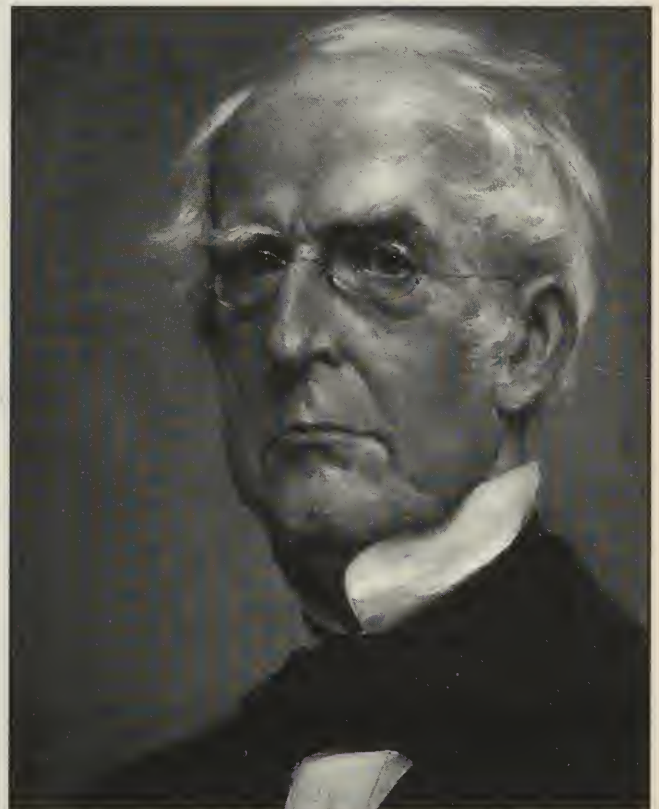


Figure 1. Portrait of Nathan Ryno Smith, M.D. 1797-1877. From the collection of the Medical and Chirurgical Faculty of Maryland.



Figure 2. Lithograph of the appearance of N.R. Smith's patient, Mrs. Wells of Prince George County. The tumor "occupied the whole space between the os hyoides above, and the sternum below . . . the skin covering its most dependent point had ulcerated . . . there had now issued . . . a dark fungous excrecence."

of swallowing, ulceration with malodor and bleeding, or simple embarrassment suffered by the victims of enormous neck masses. The courage of the surgeons was exceeded only by that of their patients, whose misery prompted them to submit to operations during the long era which preceded the availability of general anesthesia.

From the earliest times through the mid-nineteenth century, his meticulous literature search uncovered an additional 65 cases which had escaped citation in previous compilations. Among the cases was the 1835 reported case of Nathan Ryno Smith, M.D., professor of surgery at the University of Maryland from 1827 through 1867.²

Dr. Smith came to an academic career in surgery, in part through the influence of his father, Nathan Smith, M.D.,

founder of the medical schools of Dartmouth, Yale, Bowdoin, and the University of Vermont. Father of ten children, four of whom became physicians and leaders in due course, his academic legacy was passed to Nathan Ryno, born May 21, 1797.

Dr. Ryno Smith's higher education was received in New Haven, where his father was summoned in 1814 to organize the Yale Medical School after successfully launching the Dartmouth experiment sixteen years before. It was at Yale, in 1823, that Dr. Smith earned his medical degree. Subsequent to a brief stay at the University of Vermont, where he assisted his father in the organization of that medical school and served as professor of anatomy and physiology, he relocated to Philadelphia to train in surgery with Philip Syng Physick. After this experience, he briefly assisted in the inauguration of the Thomas Jefferson Medical School while serving there as professor of surgery. His stay at that institution was also transitory, due mainly to the enticement of an offer from the University of Maryland, a medical school already functioning successfully for two decades. Dr. Smith had already acquired a fondness for the South from earlier travels, so he eagerly accepted, in 1827, first the chair in anatomy and then, when it suddenly became available by the abrupt departure of Granville Sharp Pattison, the chair in surgery.

There he found his niche — teaching, operating, pioneering in the development of new operative procedures as well as the invention of unique instrumentation. He wrote and published both scientific and literary treatises. He served as dean of the School of Medicine and president of the Medical and Chirurgical Faculty of Maryland. In the days preceding the Civil War, he was an outspoken supporter of the rebellion. With his wife, Juliette Octavia Penniman Smith, they raised eight children, and their four sons became physicians. Because of his impressive physical stature, imperious demeanor, and incredible self-confidence, he became known at home and abroad as "The Emperor." His reign lasted forty years before he retired. During his career, his talents in the operating room were widely recognized and his innovative surgical procedures acclaimed. "One of the greatest surgeons America has produced . . .," claimed Professor Samuel D. Gross, himself a giant in the history of American surgery and a pupil of Dr. Smith during his brief tenure at Jefferson Medical School.

Dr. Smith is best known for his operations to cure groin hernia, remove bladder stones, relieve brain abscesses and

depressed skull fractures, and for the ligation of major arteries and veins for injury or disease. He devised numerous orthopedic operations and manipulative procedures. He operated for ovarian cyst and for scrotal hydrocele. He deftly operated upon the parotid for tumor and upon the lachrymal duct for stricture. But early in his career, he undertook an operation for massive, ulcerating goiter of the thyroid gland. By 1835, none in Maryland had previously attempted to excise a tumor of the thyroid, and only once or twice had it been attempted in the United States.

The patient, a 40-year-old woman, presented with a 20-year history of an increasingly enlarging mass of the lower anterior neck that had, in its dependent portion, breached the overlying skin. After having first rejected the notion of operating upon the woman, she finally prevailed upon him. Without anesthesia, this fearless and desperate patient underwent a protracted operative removal of this enormous tumor. Dr. Smith, in his exceedingly detailed report, states: "Twice or thrice, at her desire, I had delayed a few minutes to allow her a moment of comparative repose . . ." Neither surgeon nor patient were rewarded for their bravery, for on the thirteenth postoperative day, she expired from overwhelming sepsis.

Eighty-five years later, Dr. Halsted, professor of surgery at Johns Hopkins University, who brought to the world the technique of meticulous, bloodless, and anatomic dissection of the thyroid gland, in his landmark monograph entitled the

Operative Story of Goitre, The Author's Operation, paid homage to his distinguished predecessor.

Nathan R. Smith had quite surely never seen an operation performed upon the thyroid gland, and it is not unlikely that he had never heard of such an operation, although he concludes his paper with the sentence, "Instances in which the operation has been successfully accomplished are no doubt fresh in the minds of the readers."

My admiration for Dr. Smith, Baltimore's "Emperor," has been greatly increased since reading this modest and lucid report of a case, the importance of which he could have hardly comprehended.

On the 200th anniversary of the birth of Nathan Ryno Smith, we recognize his enormous contributions to the history of American medical education, the early development of modern surgical technique, and to the everlasting glory of his adopted state of Maryland.

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RONALD H. FISHBEIN, M.D.

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NEW PUBLICATION FOR IMMUNOCOMPROMISED WOMEN ▼ ▼ ▼ ▼ ▼ ▼



▼ A new publication for immunocompromised women, *Demi Mondaine*, was launched in July by the International Association of Physicians in AIDS Care (IAPAC). The launch issue was sent to approximately 25,000 physicians, clinics, and community organizations that provide care and services to immunocompromised women.



▼ Each issue of the magazine will feature articles on clinical topics, an advocacy topic, conference reports, and profiles of women who have successfully managed and adjusted to their immunocompromised status. These articles will be supplemented by book reviews, articles on lifestyle issues, and political action information.



▼ The magazine will be distributed free of charge to women through physicians and organizations and will also be available by subscription. For information on obtaining a copy of the July 1997 issue, write to the International Association of Physicians in AIDS Care, 225 W. Washington St., Ste. 2200, Chicago, IL 60606; Fax: 312-419-7079; E-mail: iapac@iapac.org.

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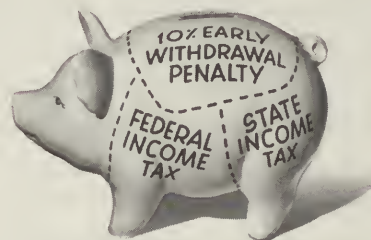
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KAISER PERMANENTE

FROM THE BPQA

The opinions expressed in this vignette are those of Dr. Winchell and have not been endorsed by the Board of Physician Quality Assurance. Dr. Winchell recused from all deliberations in this case because of a conflict of interest.

Debbie Plays Doctor

The Board of Physician Quality Assurance (BPQA) received a query from a patient whose obstetrician was in solo practice. The doctor's assistant, "Debbie," would often see the patient for her prenatal visits when the obstetrician was called away from the office to attend a delivery. On these occasions, Debbie would conduct the entire exam, including pelvic examinations when indicated. Occasionally, Debbie performed sonograms to establish fetal position or gestational age. The patient didn't have any complaints about Debbie's care; she just wanted to know what qualifications Debbie had to substitute for the doctor in this way.

The BPQA was also interested in Debbie's credentials. Debbie was interviewed and confirmed what the patient had said about her duties in the obstetrician's office. Debbie was very interested in obstetrics. Although her college degree was in liberal arts, she was seriously considering pursuing a career as a midwife. Debbie had not trained as a nurse or as a physician's assistant. She had learned her skills "on the job." From all indications, she was pretty good at what she did, and the patients had no complaints about seeing her rather than the obstetrician when necessary.

■ What would you do if you were a member of the Board of Physician Quality Assurance and this set of facts was presented to you?

The BPQA charged the obstetrician with practicing medicine with an

unauthorized person. The physician negotiated a settlement with the BPQA, which included a fine of \$20,000, three years probation, and a requirement that the training and certification of any individuals employed in the office be documented. The doctor was informed of the possibility of criminal prosecution for abetting an unlicensed individual in the practice of medicine. Debbie's activities were reported to the Board of Nursing for action. Precipitously, Debbie moved out of state to pursue her dream of becoming a nurse midwife.

Discussion by Dr. Cheryl Winchell, member of the Board of Physician Quality Assurance:

I was very distressed by this case because the obstetrician is both a fine doctor and a fine person. I wondered why the physician had acted so imprudently. Was it just to save money? Surely, Debbie didn't command the salary of a nurse midwife, a physician's assistant, or a nurse practitioner, all of whom might legitimately have functioned in the same capacity Debbie had, albeit *legally*. However, I don't think money was the motive. I believe this physician didn't perceive there was any wrongdoing. The physician had personally trained Debbie and knew what she could do competently. With the physician next door at the hospital, how could this be a problem?

Most physicians, when they hear the facts of this case, are pretty horri-

fied. But a significant minority want to know, if Debbie provided good care in the physician's absence, what is the problem? No one was hurt. There was no allegation of substandard care. There wasn't even a complaint, just a query to the BPQA. So, why did the BPQA sanction the physician?

A lot of people operate on the "no harm—no foul" principle. They don't realize we live in a society of rules. One of the rules is that in order to practice medicine, you're supposed to go to medical school. Then, you're supposed to take further training, pass a licensing examination, apply for licensure, pay a fee, and then—you can practice medicine. Debbie skipped all of the above steps. Further, she hadn't trained as a nurse. She hadn't trained as a physician's assistant. Her degree was in art history.

When this obstetrician appeared before a committee of the BPQA for termination of probation, meticulous compliance with the consent order was apparent. A member of the case resolution conference spoke in glowing terms about the record keeping and documentation, attesting that only individuals appropriately certified or licensed are currently employed. But, on numerous occasions, physicians have related to me that the obstetrician is very bitter about the disciplinary action taken by the Board of Physician Quality Assurance and has nothing good to say about the Board. Apparently, we changed the doctor's behavior, but we did not change the doctor's opinion about the appropriateness of Debbie's former job responsibilities or the BPQA's right to exact a severe sanction. No harm—no foul? ■

Accidental Falls: Their Causes and Their Injuries
Alvin S. Hyde, Ph.D., M.D. Tallahassee, FL: Rose Printing Co., Inc. 251 pages.
\$127 (hardcover), \$77 (softcover).

The author is a retired family and emergency room physician. He practiced the latter specialty for his last 15 years in medicine in Ohio, Virginia, and Maryland. He wrote this book “for readers of diverse interests, training, and experience.”

It is difficult to believe that most physicians would read this full text. However, the author appears to have anticipated this by providing a very adequate summary at the end of each chapter. One can obtain a reasonable insight into the volume's contents by reading the summaries of the first seven chapters and the entirety of chapters eight and ten — "Environmental Factors that Cause Falls" and "On Preventing Falls and Fall Injuries." While

The author makes an interesting observation — there should be better teaching in architectural schools to inculcate the need for safety in design of buildings. He seems to have little to suggest for the practicing physician in ameliorating this problem. He does suggest more attention and better supervision for the elderly hospitalized for falls. Too many of them succumb to head injuries, pneumonia, and pulmonary embolism in the first two weeks after hospitalization.

He clearly indicates the unknowns and our weaknesses but describes various studies in progress which will hopefully offer us more positive preventive and therapeutic options.

All practicing physicians, especially geriatricians, must be interested in diminishing the adverse effects of falls. This book is obviously well researched. If the reader limits his or her reading to the sections described above, it should be considered time well spent.

ER is a top-rated television program created by a well-known novelist and medical school graduate, Michael Crichton. As a medical show, it has generated much interest. What do you think of it? We would like to have your comments and critique. Send them to *MMJ* Reviews, 1211 Cathedral Street, Baltimore, MD 21201.

EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

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August, 1997

Selected Communicable Diseases in Maryland in 1996

(Continued from May/June, and July, 1997)

Please refer to the May/June issue for a description of communicable disease surveillance in Maryland and for Tables 1a and 1b, "Cases of Selected Notifiable Diseases Reported in Maryland in 1996 by County." All incidence rates in this article are expressed as cases per 100,000 population per year. All rate maps place cases by county of residence at the time of diagnosis. The spot maps place cases within their *zipcode* of residence, but with *county* boundaries displayed on those maps.

ANIMAL RABIES (637)

September 1981 marks the beginning of a raccoon rabies epizootic in Maryland; since then, cases have been confirmed in all 23 counties and Baltimore City. Figure 15 shows the trend for the past 15 years. In 1996, animal rabies was reported from all Maryland jurisdictions (Figure 16).

In 1996, 637 of the 5,504 animals submitted for laboratory examination were positive for rabies. This is a 31% increase from the 442 reported positive in 1995.

Raccoons continue to be the most frequent laboratory confirmed rabid animal (512, 80%). However, other wild, domestic, and exotic animals can be a source of rabies infection. Spillover species reported with rabies are: skunk (44, 7 %), fox (34, 5%), cat (19, 3 %), groundhog (12, 2%), cattle (2, <1%), and dog, otter, and sheep (1 each, <1%).

Bat rabies, unrelated to the raccoon epizootic, accounted for 11 (2 %) of the positive animals. The last human case of rabies reported in Maryland resulted from a bat bite in 1976.

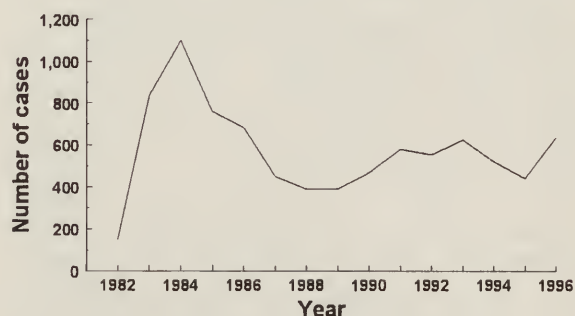


Figure 15. Animal Rabies. Cases reported, Maryland, 1982 to 1996.

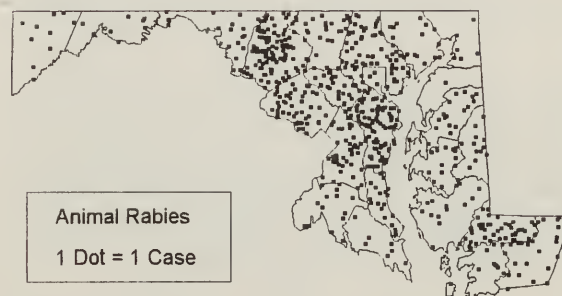


Figure 16. Animal Rabies. Maryland, 1996.

ROCKY MTN. SPOTTED FEVER (38) 0.7/100,000 (U.S. 0.3/100,000)

Thirty-eight laboratory confirmed cases of RMSF were reported in 1996. The incidence rate (0.7) is unchanged from last year. Cases were reported from 12 counties (Figure 17). The highest incidence rates were reported from Saint Mary's County (19.4) and Dorchester County (6.6).

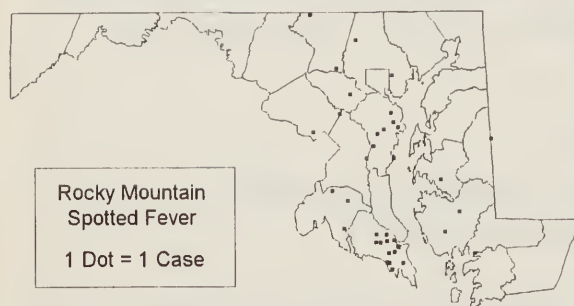


Figure 17. Rocky Mountain Spotted Fever. Maryland, 1996.

Onsets occurred between February and November. Twenty-nine cases (76%) had onsets between May and September. June was the peak month with 11 cases (29%). The age of cases ranged from 2 to 70 years (median 41 years). The ratio of male to female cases was 1.1:1. Thirty-five cases were white, 2 were black, and 1 was Asian. Ten cases (26%) were hospitalized and no deaths were reported. Exposure information was available for 33 cases; 18 reported a tick bite or attachment within 14 days of onset and 1 reported laboratory exposure to *R. rickettsii*. Information on symptoms was incomplete.

It should be noted that a number of reported cases met the case definition having had single IFA titers of 1:64 or 1:128 and relatively mild clinical signs and symptoms. This may account for the high incidence rate of RMSF in Saint Mary's County. A few clinically compatible cases were reported that did not meet the CDC case definition and were not counted because they were not laboratory confirmed (convalescent titers were not collected).

SALMONELLOSIS (1160) 22.8/100,000 (U.S. 16.7/100,000)

The number of cases reported by jurisdiction in 1996 is shown in Table 1a. Statewide totals for the past ten years are shown in Figure 18. The trend seems to have stabilized over the past six years, with approximately 1,000 to 1,300 cases reported per year. Incidence of disease in 1996 was highest in the warmer months, with 38% of onsets occurring from June through September. Salmonellosis rates by jurisdiction are shown in Figure 19. The highest rates were observed in Somerset (85.6), Caroline (58.0), and Dorchester (55.9) Counties.

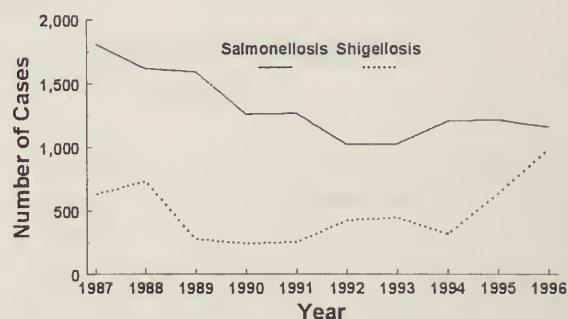


Figure 18. Salmonellosis and Shigellosis. Cases reported, Maryland, 1987-1996.

The male to female ratio was 1:1. The ratio of whites to non-whites was 1.5:1 (16% had unknown race). The highest rate of infection by five year age groups was in the birth to 4 years group (97.7), followed by children 5-9 years old (27.9). In adults the highest rate was observed in the 20-29 years age group (17.5). There were 296 hospitalizations for salmonellosis and 10 deaths.

Of the 730 (63%) isolates for which serotype information was available, 308 (42%) were *S. Enteritidis*. The four other most frequently reported serotypes were: *S. Typhimurium* (175, 24%), *S. Newport* (37, 5%), *S. Muenchen* (33, 5%), and *S. Heidelberg* (26, 4%).

There were eight outbreaks of salmonellosis reported by four counties. Seven of the outbreaks resulted from foodborne transmission. Four of the outbreaks occurred in restaurants, three in private homes, and one in a migrant labor camp.

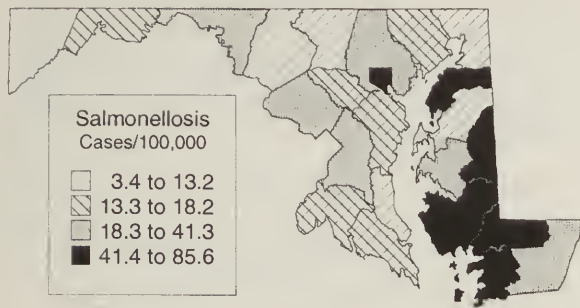


Figure 19. Salmonellosis. Maryland, 1996.

SHIGELLOSIS (985) 19.3/100,000 (U.S. 9.1/100,000)

Maryland reported 985 cases of culture confirmed shigellosis in 1996, a sharp increase (54%) from the 639 cases reported in 1995 (Figure 18). This represents the second year of such an increase. The number of cases by jurisdiction is shown in Table 1a. Sixty-nine percent of the cases occurred in Prince George's County, Baltimore City, Wicomico County, and Montgomery County, which account for 47% of Maryland's population. Wicomico County reported the highest rate (152.1), followed by Dorchester County (75.6), Somerset County (44.8), and Kent County (42.6) (Figure 20).

The ratio of male to female cases was 0.9:1. The ratio of cases among whites compared to non-whites was 0.6:1; the race of 15% was unknown. Most cases occurred among young children. The highest annual rates occurred in the birth to 4 years age group (88.0) and the 5-9 years age group (62.0), with a total of 564 cases (57%) of shigellosis occurring among children under the age of 10. There were 108 hospitalizations and two deaths reported.

Of the 946 (96%) isolates for which the species was known, 885 (93.6%) were *S. sonnei*, 56 (5.9%) were *S. flexneri*, 3 (0.3%) were *S. boydii*, and 2 (0.2%) were *S. dysenteriae*. The one case of *S. dysenteriae* that was available for interview had no history of travel outside the United States. The majority (86%) of the *Shigella* isolates tested by the DHMH laboratory have been resistant to ampicillin. Most isolates however, have been found to be susceptible to trimethoprim-sulfamethoxazole, cephalosporins, and quinolones.

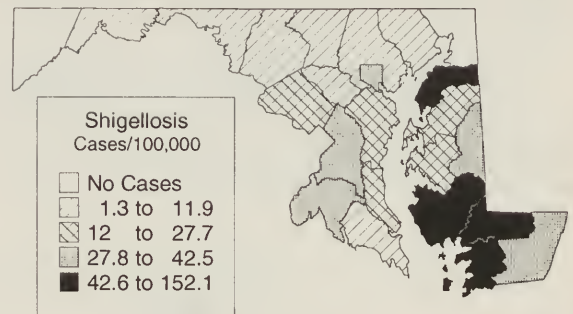


Figure 20. Shigellosis. Maryland, 1996.

In 1996, a total of 15 outbreaks of shigellosis were reported from 11 jurisdictions. The outbreaks occurred in 9 day care centers, 3 restaurants, a private home, a community, and in a group that traveled to Peru.

TUBERCULOSIS (319) 6.3/100,000 (U.S. 7.2/100,000)

The number of reported tuberculosis cases declined 14% from 1995 (370). The trend of TB case reporting since 1982 is shown in Figure 21. The number of cases reported by jurisdiction in

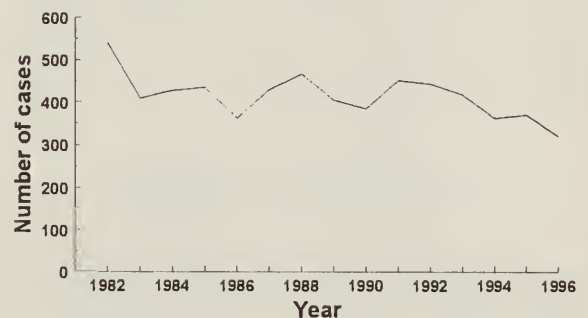


Figure 21. Tuberculosis. Cases reported, Maryland, 1982-1996.

1996 is shown in Table 1a, as is the State total for the previous five years. Baltimore City, Prince George's and Montgomery Counties reported 32%, 19%, and 16%, respectively, of all cases in 1996 (Figure 22). The ratio of male to female cases was 1.3:1. Rates were highest among Asians (43), followed by blacks (14.2), Hispanic (9.6), and white (2.3). Thirty-six percent (114/319) of all cases were foreign-born. Rates increased progressively with increasing age, with the lowest among children

under 14 years (1.5) and the highest rates occurring in those 65 years and older (17.8).

A match of the tuberculosis and AIDS registries for 1996 identified 33 (10.3%) persons with both diseases, compared to 30 (8.1%) in 1995, 39 (11%) in 1994, 41 (10%) in 1993, and 60 (14%) in 1992.

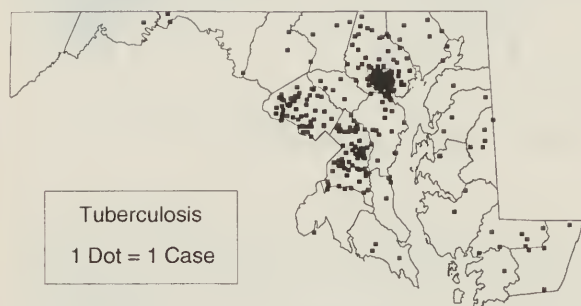


Figure 22. Tuberculosis. Maryland, 1996.

Drug resistance has not been a significant problem in Maryland. In 1996, only 15 (5%) of the reported cases were isoniazid (INH) resistant, 3 (0.9%) rifampin resistant, and 1 (0.3%) was resistant to both INH and rifampin.

Directly Observed Therapy (DOT) (the observation by trained health care workers of every dose of medication taken) is still a high priority public health strategy in Maryland and is considered the standard of care for TB patients. In 1996, 90% of TB patients received DOT.

TYPHOID FEVER (18) 0.4/100,000 (U.S. 0.1 /100,000)

There was a three-fold increase in reported acute typhoid fever from 1995 to 1996. Cases were reported from Montgomery (8), Baltimore (3), Garrett (1), Harford (2), Howard (2), Prince George's (1), and Wicomico (1) Counties.

The ratio of male to female cases was 1.3:1. Seven cases were Asian, 3 were white, 2 were black, 3 were "other" races; the race of 3 cases was unknown. Ages ranged from 11 months to 48 years (median 9.5 years). There were 16 hospitalizations, but no deaths occurred.

International travel accounted for 14 of the 18 cases; the countries visited were India (5), Bangladesh (1), Pakistan (3), Cambodia (1), El Salvador (1), Nigeria (1), Sierra Leone (1), and

Vietnam (1). Two additional cases occurred in household contacts of a recent traveler to Bangladesh who could not be confirmed as a case. Two cases reported no foreign travel, and no source could be identified.

SEXUALLY TRANSMITTED DISEASES

CHLAMYDIAL INFECTIONS (20,705) 407/100,000 (U.S. 147/100,000)

Chlamydia trachomatis became a reportable condition by laboratories in October, 1994. This is the first year that it is being included in this summary.

Eighty-three percent of the 20,705 cases reported in 1996 were in the 15-29 years age group. Women represented 82% of the reported cases; this can be attributed to the fact that women are screened routinely in family planning, STD, and maternity settings. Jurisdictions with rates exceeding 300 cases per 100,000 population are: Baltimore City (1,969), Somerset (453), Wicomico (429), Worcester (421), Caroline (379), and Prince George's (328) counties. Figure 23 shows the distribution of *Chlamydia* rates in Maryland in 1996.

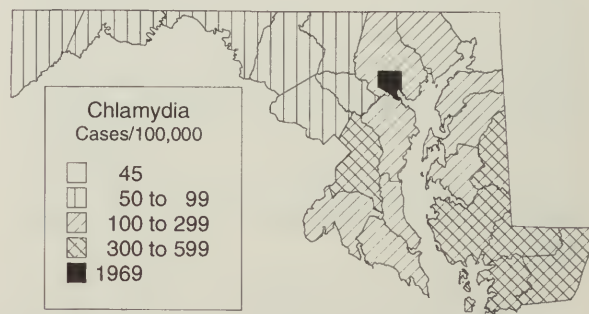


Figure 23. Chlamydial infection. Maryland, 1996.

GNOCOCCAL INFECTIONS (11,316) 222/100,000 (U.S. 116/100,000)

Reported gonococcal infections decreased 23 percent from 1995. Since 1990, reported cases have declined 52 percent. Maryland and U.S. incidence rates for the last 10 years are shown in Figure 24.

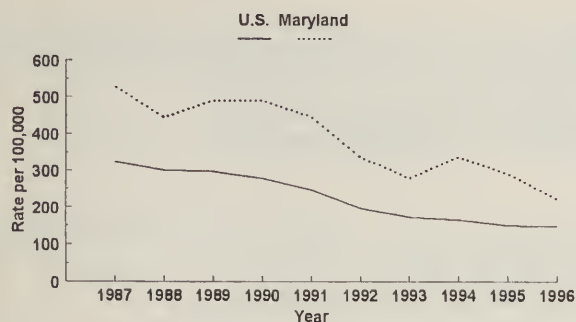


Figure 24. Gonococcal infection. Maryland and the United States, 1987-1996.

Baltimore City noted a 28% decline since 1995; the counties, excluding Baltimore City, reported a decrease of 16%. The number of cases by jurisdiction is shown in Table 1a. Fifty-six percent of the cases were in Baltimore City residents, who also accounted for the highest rate (911) in the State. Counties with the highest rates were: Dorchester (529), Wicomico (444), Somerset (330) (Figure 25).

The age distribution of the cases was similar to previous years: 80% of the cases were reported in the 15-34 years age group.

SYPHILIS, PRIMARY AND SECONDARY (733) 14.4/100,000 (U.S. 4.2/100,000)

In 1996, Maryland reported 733 cases of primary and secondary (P&S) syphilis; this is a correction from the 808 cases reported in Table 1a. The 733 cases represents a 29% increase from the 567 cases reported in 1995. Figure 26 shows the trend of P&S incidence in Maryland over the past 10 years. The number of cases in 1996 by jurisdiction is presented in Table 1a (Baltimore City

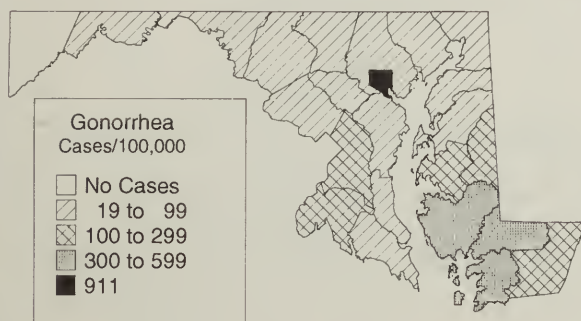


Figure 25. Gonococcal infection. Maryland, 1996.

should be corrected to 557 cases). For the second consecutive year, Baltimore City was primarily responsible for the observed increase, and accounted for 76% of all case reports in Maryland.

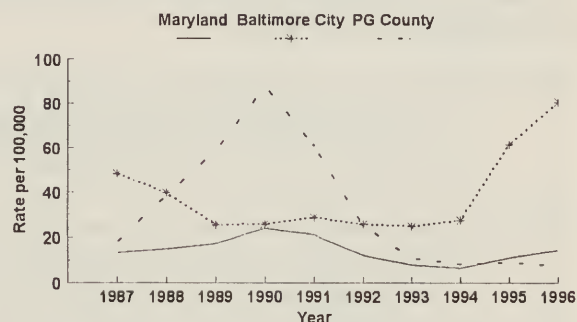


Figure 26. Primary and secondary syphilis. Incidence, Maryland, 1987-1996.

The ratio of male to female cases was 1.3 to 1. The race of 92% of the cases was specified as non-white. Sixty-six percent of the cases were in the 20-39 year age group.

Since 1988 all P&S and early latent syphilis cases seen in public STD clinics have been offered HIV testing. The percent of co-infection in those tested has decreased each year, from 18% (66/372) in 1988 to 6% (55/920) in 1996.

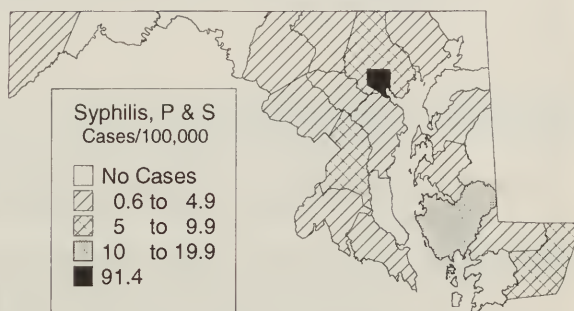


Figure 27. Primary and secondary syphilis. Maryland, 1996.

The number of congenital syphilis cases (45) increased 88% from 1995 (24). The following jurisdictions reported cases: Baltimore City (34), Prince George's County (4), Baltimore County (4) and one each in Anne Arundel, Carroll and Montgomery Counties.

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

- | | |
|---|--------------------|
| 25th annual geriatrics symposium: Current topics in geriatrics , sponsored by the Johns Hopkins Geriatrics Center, at the Renaissance Harborplace Hotel, Baltimore. Credits: 19 Cat 1 AMA credits. Fee: \$400/physicians; \$300/residents, fellows, allied health professionals. | Aug. 21–23 |
| 26th annual diagnostic ultrasound in gynecology and obstetrics and abdomen , sponsored by the Johns Hopkins department of radiology and radiological science, at the Renaissance Harborplace Hotel, Baltimore. Credits: TBD. | Sept. 5–7 |
| Fifth annual progress in hematologic malignancies and bone marrow transplantation , sponsored by the division of hematologic malignancies, department of oncology, Johns Hopkins. Credits: 7.5 Cat 1 AMA credits. Fee: \$100/alumni past registrants; \$125/new registrants. | Sept. 19 |
| 23rd annual topics in gastroenterology and liver disease , sponsored by the Meyerhoff Center for Digestive Disease. Credits: 24 Cat 1 AMA credits. Fee: \$535/physicians; \$285/residents, fellows. If postmarked prior to August 1: \$495/physicians; \$250/residents, fellows. | Sept. 24–26 |
| 39th annual Emil Novak memorial course: Gynecology, gynecological pathology, endocrinology, and high risk obstetrics , sponsored by the Johns Hopkins department of obstetrics and gynecology, at the Hyatt Regency Inner Harbor Hotel, Baltimore. Credits: 45.5 Cat 1 AMA credits. Fee: \$950/physicians; \$750/residents, fellows. | Oct. 5–10 |
| Advanced pediatric life support courses , sponsored by the Johns Hopkins University School of Medicine and the Johns Hopkins Pediatric Trauma Center. Credits: 21 Cat 1 AMA credits. Fee: \$595. | Oct. 27–29 |

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Self-Directed CME Activities

CD-ROM-based interactive multimedia radiology teaching file for Mac or PC w/single user licenses (SUL), site licenses (SL), or multisite licenses (MSL). Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

- | | |
|---|------------------------|
| Clinical breast examination using MammaCare technique: A practicum for physicians , sponsored by the Medical and Chirurgical Faculty of Maryland, at Union Hospital, Elkton, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056. | Aug. 20 |
| Neuroradiology review: Including the head, neck and spine , sponsored by the University of California, Irvine, at The Four Seasons Hotel, Newport Beach, CA. Credits: 28 Cat 1 AMA credits. Fee: \$725/physicians; \$450/residents, fellows, technologists, retired physicians. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Aug. 31–Sept. 4 |
| Clinical breast examination using MammaCare technique: A practicum for physicians , sponsored by the Medical and Chirurgical Faculty of Maryland, at Kent and Queen Anne's Hospital, Chestertown, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056. | Sept. 3 |
| The International Skeletal Society 24th annual refresher course , sponsored by The International Skeletal Society, at The Sweeney Convention Center, Santa Fe, New Mexico. Credits: 24.5 Cat 1 AMA credits. Fee: \$650/physicians; \$425/residents, fellows, technologists, military personnel, retired physicians. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Sept. 10–13 |
| Mount Sinai 1997 update: Brain, spine, neurovascular & ENT imaging , sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education, at The Plaza Hotel, New York, New York. Credits: 25.5 Cat 1 AMA credits. Fee: \$675/physicians; \$375/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). | Sept. 10–14 |
| Immunization update 1997 , presented via satellite by the Centers for Disease Control and Prevention, hosted by the Center for Immunization, Maryland Department of Health & Mental Hygiene. Credits: CMEs available. Fee: none. Info: Sandra Kash, 410-767-6679. | Sept. 11 |
| Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Carroll County General Hospital, Carroll County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. | Sept. 12 |

Miscellaneous (continued)

Organ imaging review 1997, sponsored by The University of Toronto department of medical imaging, at The Toronto Hilton, Toronto, Ontario, Canada. Credits: 28 Cat 1 AMA credits. Fee: \$520/physicians; \$370/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Sept. 14-18**

The revolution in diabetes: Prevention and treatment, sponsored by Adventist HealthCare System at Martin's Crosswinds, Greenbelt, Maryland. Credits: 7 Cat 1 AMA credits. Fee: \$110 (before Aug. 22); \$120 (after Aug. 22). Info: 301-891-5621. **Sept. 18**



PHYSICIAN'S RECOGNITION AWARD

From July 1996 through November 1996, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. They were mistakenly not included in the *Maryland Medical Journal* Physician's Recognition Award listing during this time. We apologize for the omission.

Peter J. Bianchine
Stewart J. Callis
Venkataraman P. Chandar
Joseph H. Cutchin
Kathleen K. Davis
Jonathan Gitter
Allan R. Glass
David Green

Larry B. Grossman
Wendelin S. Hayes
Barbara K. Honig
Noel S. Howard
Daniel M. Howell
Irwin H. Marill
Brian P. Monahan
Ronald S. Mukamal

Felicia M. Ollivierre
Otto Roza
Michael A. Schwartz
Hamid Towhidian
Lewis R. Townsend
Stuart J. Turkewitz
Paul F. Vietz
Nieves M. Zaldivar

During April 1997, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

Susanne R.M. Adamson
Susan E. Bailey
Robert S. Berger
Rohit Bhatnagar
James F. Carow
Manuel B. Datiles
Nabil A. El-Shammaa
Arman K. Fard
Hugo E. Gallo-Torres
Jose A. Gelpi
Carol M. Gonzalez
Ramon L. Gonzalez

Ching-Jou Gou
David H. Grossman
Donald R. Haggerty
Chester Z. Haverback
William C. Hewitson
Yong Hyen Hwang
William J. Jaffurs
Leeds E. Katzen
Sitaramamma Kottapalli
Raul A. Lazarte
Iradj Mahdavi
Rekha P. Motagi

Jafar Nazemian
Michael M. Raffinan
Michael A. Randolph
Alice P. Rieckelman
Alfred B. Rosenstein
Anne M. Shewan
Irene G. Tamagna
Joseph R. Tiralla
Amanda C. Trimpey
Mario Vahos
Jeffrey L. Warhaftig

Miscellaneous (continued)

- Clinical breast examination using MammaCare technique: A practicum for physicians,** Sept. 19
sponsored by the Medical and Chirurgical Faculty of Maryland, at Peninsula Regional Medical Center, Salisbury, MD. Credits: 2 Cat 1 AMA credits. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.
- Cancer prevention in community practice,** Sept. 24
sponsored by the Medical and Chirurgical Faculty of Maryland, at Harbor Hospital, Baltimore City. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.
- Eighth international symposium on diagnostic imaging: State of the art on thoracic diseases,** Oct. 2-4
sponsored by The University of Barcelona and The University of Florida, College of Medicine, at the Hotel Arts Barcelona, Spain. Credits: 18 Cat 1 AMA credits. Fee: \$350/physicians; \$250/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).
- Cancer prevention in community practice,** Oct. 8
sponsored by the Medical and Chirurgical Faculty of Maryland, at Washington County Hospital Association, Washington County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.
- Infectious disease '97 board review: A comprehensive review for board preparation,** Oct. 15-19
sponsored by The Center for Bio-Medical Communication, at the Ritz-Carlton, Tysons Corner, McLean, Virginia. Credits: 40 Cat 1 AMA credits. Fee: \$895/physicians; \$695/physicians-in-training. Info: 201-385-8080, Fax 201-385-5650 (e-mail: cbcbiomed@aol.com).
- Neuroradiology update,** Oct. 16-17
sponsored by The University of California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, California. Credits: 13 Cat 1 AMA credits. Fee: \$300/physicians; \$200/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).

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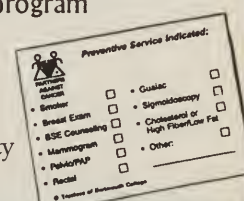
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The Medical and Chirurgical Faculty of Maryland is currently offering this program at the following locations/dates:

- ◆ September 12, 1997 at Carroll County General Hospital, Carroll County
- ◆ September 24, 1997 at Harbor Hospital, Baltimore City
- ◆ October 8, 1997 at Washington County Hospital Association, Washington County
- ◆ October 29, 1997 at Memorial Hospital at Easton, Talbot County



Call Carol Schwartz, Project Coordinator at: 1-800-492-1056 or 410-539-0872 to register or inquire further.

Miscellaneous (continued)

- New techniques in urinary incontinence and female urology**, sponsored by The Washington University School of Medicine, at the Eric P. Newman Education Center, Washinton University Medical Center, St. Louis, Missouri. Credits: 8.5 Cat I AMA credits. Fee: \$200/physicians; \$100/physicians-in-training. Info: 800-325-9862, Fax: 314-362-1087. **Oct. 18**
- UCSD postgraduate radiology course: Musculoskeletal, obstetrical, and body imaging, neuroradiology, chest imaging, and women's imaging and pediatrics**, sponsored by The University of California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, California. Credits: 40 Cat I AMA credits. Fee: \$1,000/physicians; \$700/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Oct. 20-24**
- Musculoskeletal MR**, sponsored by The University of California, San Diego, School of Medicine, at the Westin Resort Hotel, Hilton Head, South Carolina. Credits: 20 Cat I AMA credits. Fee: \$550/physicians; \$375/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Oct. 22-26**
- New techniques and concepts in cardiology**, sponsored by The American College of Cardiology, at the Hyatt Regency Capitol Hill, Washington, D.C. Credits: 16 Cat I AMA credits. Info: 800-253-4636, ext. 695, Fax: 301-897-9745. **Oct. 23-25**
- Occupational and environmental medicine: Clinical practice in progress**, sponsored by the American College of Occupational and Environmental Medicine, at the Opryland Hotel in Nashville, Tennessee. Info: 847-228-6850, ext. 184, Fax: 847-228-1856. **Oct. 26-30**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Memorial Hospital at Easton, Talbot County. CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Oct. 29**

Self-Directed CME Activities

- Maryland physicians' campaign against family violence, module one: Domestic violence**, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat I AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.
- Maryland physicians' campaign against family violence, module two: Child maltreatment**, sponsored by the Medical and Chirurgical Faculty of Maryland and the Maryland Alliance Against Family Violence. Credits: 4 Cat I AMA credits. Fee: \$10. Info: 410-539-0872. Expires September 5, 1997.

Continuously throughout the year

- Fluorescein angiography conference**, sponsored by the Retina Center of Maryland, Baltimore, MD. First and third Mondays of each month, 8:00 a.m. - 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.
- Sinai Hospital of Baltimore medical grand rounds**, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.

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*For more information, call Vivian Smith at
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Reserve your hotel room now by calling the Sheraton at 800-638-2100. Request Med Chi group rates.

Questions? Call Med Chi's Department of Education and Information at 410-539-0872 or 800-492-1056.

Sheraton Fontainebleu
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September 5-7, 1997

Preliminary Schedule

Friday, September 5, 1997

A.M.: House of Delegates
American Heart Association presentation
End of Life Care
Handling Drug Abusing Patients

P.M.: Reference Committees
Telemedicine
Breast Cancer and Prostate Cancer Screening Updates
Antibiotic Resistance

Evening: WELCOME BARBECUE ON THE BEACH

Saturday, September 6, 1997

A.M.: Exhibits open all day
Plenary Session: Clinical and Practice Tactics
Maryland Society of Anesthesiologists presentation

P.M.: House of Delegates
Risk Management
Internet
Good News about Osteoporosis
Asthma
AIDS

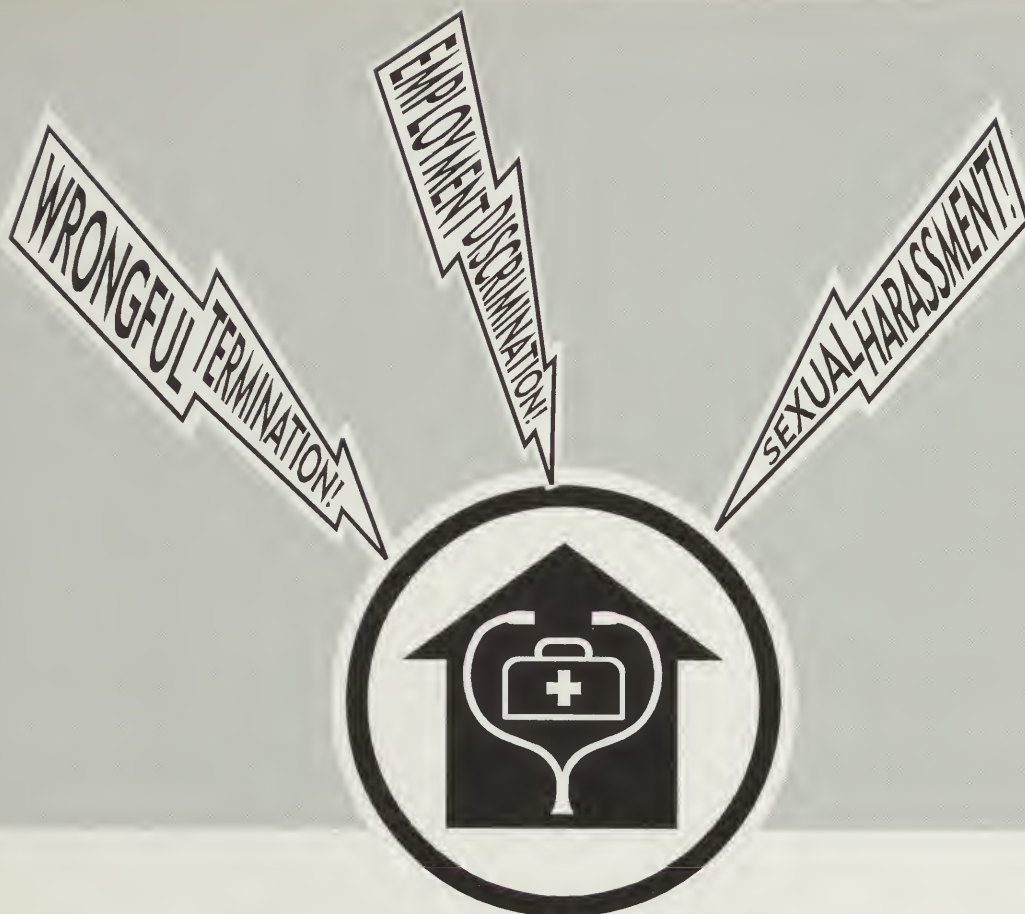
Evening: CRAB FEAST

Sunday, September 7, 1997

A.M.: Plenary Session: Med Chi Credentialing/Managed Care Contracting
Exhibits open
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Golf outing and River Run Club House brunch





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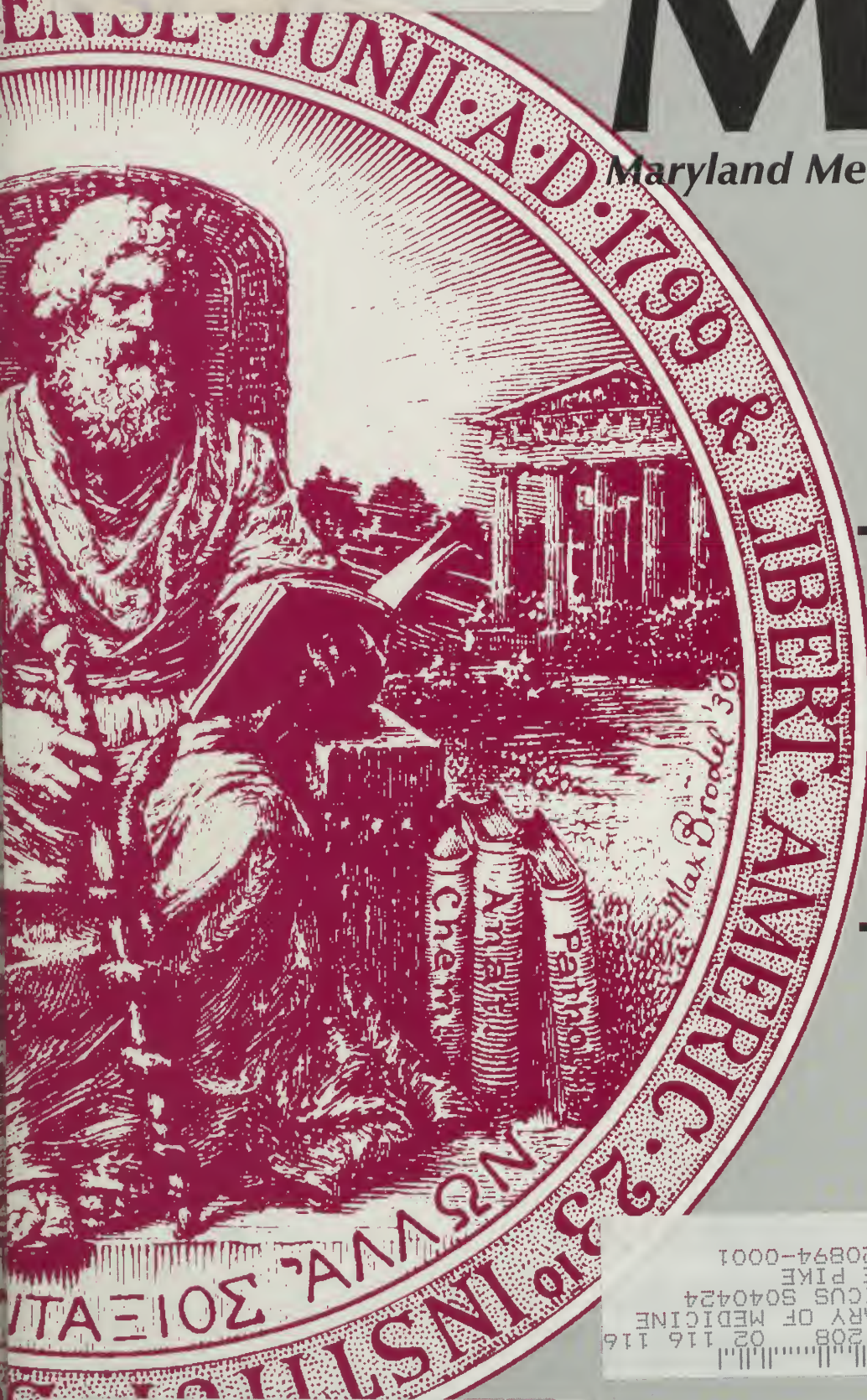
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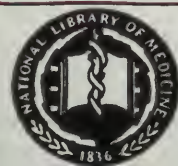
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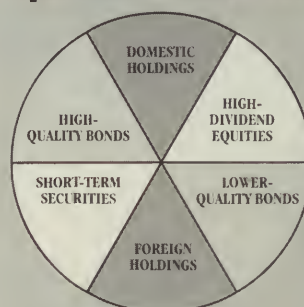
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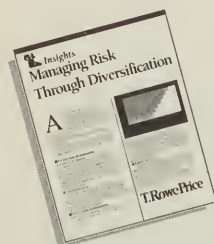
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Editor's Introduction

This issue of the Maryland Medical Journal highlights Maryland's women physicians, past and present. The guest editor is a woman who plays a prominent and important role in medicine in our state — Dr. Eve Higginbotham. Dr. Higginbotham is professor and chair of the department of ophthalmology at the University of Maryland, Maryland Center for Eye Care. She is also chair of Med Chi's Women in Medicine Committee and a member of this journal's editorial board.

Dr. Higginbotham holds two degrees in chemical engineering from Massachusetts Institute of Technology (MIT) and a medical degree from Harvard Medical School. After an internship, she completed a residency in ophthalmology at the LSU Eye Center and a fellowship in glaucoma at the Massachusetts Eye and Ear Infirmary. She has since followed an academic career in Illinois, Michigan, and now Maryland. Prior to arriving in Maryland in 1994, Dr. Higginbotham served as assistant dean for faculty affairs.

MARION FRIEDMAN, M.D.
EDITOR

Guest Editor's Introduction

Two years ago, Jane Short wrote in her article, "A Brief History of Women and Organized Medicine," "As the number of women physicians continues to grow, it is critical that organized medicine seek ways to encourage participation and leadership from women." Although women comprise more than 50% of classes entering medical schools, their numbers have not kept pace within the American Medical Association (AMA) leadership and organized medicine (Md Med J 1995;44(9):709-710). There may have been several factors contributing to this observation. As the traditional caregivers in families, women physicians may peak later in their careers than their male colleagues. Another factor may be the lack of role models or the absence of visibility of those role models who do exist. This special edition of the Maryland Medical Journal (MMJ) addresses the latter concern.

This issue was conceived by the Women in Medicine Committee and mostly written by women in medicine. Short biographies of those who contributed original articles have been included to inform aspiring young doctors about the career paths of the authors.

The first three articles highlight three areas of the medical profession — anesthesiology, primary care, and occupational medicine. The next three articles, "Glaucoma: A preventable cause of blindness," "Hormonal strategies of the menopause" and "The role of large core needle biopsy in locally advanced breast cancer" provide overviews of topics related to patient care in ophthalmology and gynecology. Doctors Zorayda Lee-Llacer and Beverly Collins provide their perspectives on socioeconomic and policy issues which confront all physicians today in their respective articles, "Managed care: Physician ownership issues" and "Review of universal health insurance: Should we try it?" Two very interesting cases, both gynecological, complete the original submissions. The diversity of the topics in this issue reflects the breadth of interests among women physicians in Med Chi.

This issue also honors three women physicians who have helped to shape the history of medicine in the state of Maryland. Doctors Celeste Woodward and Helen Taussig have made significant contributions, and their biographies are shared with us in "Maryland Medical History." A review of the memoirs of Dr. Lillian Welsh reminds us about the significant social and educational barriers that existed at the end of the nineteenth century.

Each September, the AMA celebrates the contributions of women in medicine. As guest editor, I am grateful that the editorial board supported the publication of this issue, and thus joins organized medicine in celebrating women physicians. Considering that women became members of Med Chi almost 30 years prior to the AMA, this special edition of the MMJ becomes another historical milestone in the life of the society.

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Recent developments in anesthesiology

M. Jane Matjasko, M.D.

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ABSTRACT: *This article focuses on recent developments in anesthesiology, including the expanded perioperative role of the anesthesiologist and the value these services add to an integrated delivery system by improving efficiencies and providing significant cost savings. Research activities are concerned with specific receptor mechanisms of drug action, as well as mechanisms of cell injury and post ischemia death.*

Developments in anesthesiology

Despite the reduced numbers of U.S. medical school graduates choosing anesthesiology as a career (only 253 in the 1997 NRMP Match, as compared to over 940 in 1991) and the emphasis on primary care options, there are exciting opportunities in the field of anesthesiology, adding significant value to a system of patient care. This brief article focuses on recent developments in clinical care, education, and research.

Clinical care

The anesthesiologist's services are no longer focused exclusively in the operating room. The perioperative role of the anesthesiologist is broad and includes preoperative evaluation centers, intensive care unit (ICU) and floor bed management responsibilities, and intraoperative care in academic medical centers, often as a part of a dedicated team in a variety of specialized and intensely collaborative programs (e.g., high risk obstetrics, cardiac, lung, liver transplantation, neurosurgical care, pediatric anesthesia, and perioperative pain management and critical care).^{1,2,3}

In the preoperative center, anesthesiologists, nurse practitioners, clerks, and support staff (e.g., phlebotomists, x-ray technicians) gather and

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evaluate pertinent data in preparation for ambulatory and morning admit surgery patients. More frequently now, the history, physical, and laboratory data are gathered from sources external to the system by fax or other means. Anesthesia preoperative discussion and pain management planning occur by telephone. The stable patient, and even many complex patients, do not need to be seen at the center prior to their procedure. This process assures that appropriate information is available to permit an excellent outcome of both anesthesia and surgery with minimal inconvenience to the patient. Unnecessary and repetitive lab testing is avoided. Unnecessary consultations in stable and in many severely ill, but well-managed, patients are avoided as contact with the primary care provider is essential to optimal care. This process, led by the anesthesiologist, permits more efficient and economical care and improved satisfaction for the patient, surgeon, and the anesthesiologist providing the hands-on care in the operating room. Cost savings are substantial when one considers the significantly decreased cancellation rate and the reduction, by as much as 50%, in lab tests per patient.^{4,5}

This process has met with enthusiastic approval at the University of Maryland, particularly since there is reliable direct communication between the anesthesiologist and surgeon for problem resolution. If an unavoidable barrier exists to proceeding with the case, there is ample time (>48 hours) for substitution of another patient to avoid wasting resources. The surgeon's office staff is an integral part of the success of this process since they can do direct scheduling of appointments for patients who require an in-person visit in the center (this is most often patients who require substantial preoperative teaching) and forward, via fax, all pertinent data to the center in a timely manner.

High-risk obstetric patients include those with abnormal fetal positions, multiple gestations, antepartum hemorrhage (i.e., placenta previa, abruptio placenta, uterine rupture), premature rupture of membranes, pregnancy-induced hypertension, heart disease, amniotic fluid embolism, postpartum hemorrhage, intrauterine fetal surgery, and intercurrent disease during pregnancy. Since organogenesis occurs in the first 13 weeks of gestation, it is wise to avoid anesthetic drugs in this period, although no anesthetic drug has been shown to be teratogenic. If surgery is required during pregnancy, management will be dictated by the concurrent disease and is best coordinated with the obstetrician and obstetric anesthesiologist to optimize outcome if the pregnancy is expected to continue or if a cesarean delivery is to precede the required surgery (e.g., craniotomy for aneurysm).

Cardiac transplantation is reserved for patients with end-stage heart disease who are likely to succumb in a few months. Renal and hepatic dysfunction due to chronic hypoperfusion and venous congestion must be reversible to assure graft survival. Since the cold ischemia time for cardiac grafts is short, team coordination is essential to not prolong cardio-pulmonary bypass or organ ischemia. One-year survival is in the 80% to 90% range; over 70% survive five years.

Lung transplantation is indicated in end-stage pulmonary disease for infectious, obstructive, restrictive, or vascular diseases. These procedures are done urgently to enhance graft survival. Single lung transplants are performed without cardio-pulmonary bypass and with strict attention to asepsis while placing invasive monitors as is the case in all transplants.

Liver transplantation is reserved for patients with end-stage liver disease who are no longer amenable to medical therapy. One-year survival rates exceed 85%; five-year survival exceeds 60%. Major problems for the anesthesiologist include coagulation defects, massive blood loss, the metabolic consequences of the anhepatic phase of the procedure (citrate toxicity leading to hypocalcemia and myocardial depression), and air embolism and hyperkalemia once circulation to the graft liver is established. All transplant patients described above require expert intensive care management to detect and treat complications early and to manage the sometimes tumultuous course.

Anesthesia services are less centralized in order to accommodate patient needs for invasive or non-invasive procedures in radiology, catheterization laboratories, magnetic resonance imaging suites, or endoscopy suites. This is particularly true for pediatric patients who may require brief, profound levels of anesthesia for procedures that do not require an operating room (e.g., dressing changes, drain removal). Anesthesia care is also now being delivered in physician's offices, particularly for plastic surgery, although medical and facility care standards are still being defined.

The anesthesiologist has a significant role in cost reduction.⁶ Well-recognized strategies reduce drug costs, reduce ICU bed utilization (e.g., cardiac fast-track program), maximize low-risk monitoring bed utilization, or totally eliminate the need for ICU stay. Thoracic epidural catheter placement for post thoracotomy pain management in certain patients has eliminated post-operative ventilator requirements and significantly reduced respiratory complications; no ICU time is needed.

During major neurosurgical procedures, critical neural pathways may be threatened. Motor- and sensory-evoked potential monitoring permits identification of intraoperative decrements to neural function when it can be directly remedied.⁷ Retractor adjustment, increased perfusion pressure, or position changes can be accomplished before function is irreversibly affected. Preservation of neural function clearly translates into lower costs, reduced length of stay, and improved functional outcome. Anesthesiologists are uniquely qualified to interpret the results of monitoring, particularly since anesthetic agents must be chosen that do not interfere with data collection, and because intraoperative control of the patient's blood pressure, temperature, and oxygenation, all of which can negatively and globally influence the neurophysiologic evoked potentials, are under the direct control of the anesthesiologist.

Intraoperative transesophageal echocardiography (TEE) is the standard of care during cardiac surgery, but also is of great value in patients with significant cardiac disease having non-cardiac surgery and in patients with suspected aortic injury. TEE can detect global and regional ventricular wall motion abnormalities, which are considered the earliest sign of myocardial ischemia. Intraoperative detection of flow abnormalities related to new valve implants can significantly reduce morbidity and mortality. TEE is also valuable in heart and liver transplant patients. Appropriate development of the cognitive and technical skills to perform TEE is required for optimization of this as an intraoperative tool.⁸

Education

Educational developments in the field of anesthesiology are quite unique. Several simulation centers exist throughout the country to foster skill retention.⁹ The most sophisticated center was developed in conjunction with LinkÖ, a company which developed realistic aircraft simulators that mimic real-life conditions and allow pilots to demonstrate their competence during a crisis situation. Many anesthesiology simulator centers share resources among various institutions and providers. Research is in progress to assess the impact of this type of recurrent crisis management training on the quality of patient care and the maintenance of skill levels in anesthesiologists.

Research

Research in anesthesiology is focused on the cardiorespiratory and nervous systems. Research questions are focused at cellular, subcellular, and genetic mechanisms of function and dysfunction. The central nervous system is at

risk for ischemia in a number of elective and emergent clinical circumstances that involve the anesthesiologist (e.g., open ventricle cardiac surgery, intracerebral aneurysm, intra-operative and critical care, cardiac arrest). Prevention of permanent brain damage is possible in some of these clinical circumstances. As our understanding of necrotic and apoptotic cell death expands, treatment strategies will be developed based on the understanding of these fundamental mechanisms.¹⁰

The search continues for anesthetic drugs with very specific receptor actions to permit greater drug efficacy and minimal to no side effects. For example, intrathecal and epidural opioids are widely used for pain control post surgery and for some medical conditions, such as pancreatitis and intractable angina. However, much effort is expended in managing side effects (e.g., pruritus, respiratory depression, urinary retention). New agents such as \int_2 agonists (e.g., clonidine) are efficacious in treating pain by releasing acetylcholine at the spinal cord level.¹¹ Perhaps a combination of intrathecal compounds will afford maximal pain relief with minimal side effects.

The mechanism of action of general anesthetics is elusive despite 150 years of practice. Many compounds, both inhaled and intravenous, act via specific gamma amino butyric acid (GABA) receptor subsets. Identifying the specificity of these subsets may permit the development of anesthetics that have minimal systemic trespass.¹²

Despite the concern about the number of specialist physicians, it is clear that the risks of anesthesia have declined significantly over the past decade coincident with an increased number of physician providers. During that time, there was a doubling of the number of anesthesiologists in the U.S., a significant reduction in malpractice premiums charged to anesthesiologists, and a decrease in the death rate specifically related to anesthesiology.

The American Society of Anesthesiologists (ASA) has sponsored a long-term in-depth analysis of anesthesiology closed claims (case data provided by major malpractice carriers in the United States). Case analysis has been ongoing since 1985 and has permitted focused outcomes research.¹³ For example, adverse respiratory events are extremely rare in cases where end tidal carbon dioxide monitors and pulse oximeters are in use. On the other hand, difficult airway management is less safe, and the ASA has developed clinical guidelines for difficult airway management to educate and improve performance in this area.¹⁴ These closed claims analyses have also led to basic research into the mechanisms of cardiac arrest during spinal anesthesia (not frequent, but

occurs in healthy patients)¹⁵ and for peripheral nerve injury (particularly ulnar nerve) related to the perioperative experience. Even with exquisite attention to intraoperative patient positioning, this complication occurs and its mechanism is not understood. This injury accounts for the second largest class of injury in the closed claims project. There is still much work to do to eliminate patient injury in the perioperative period and anesthesiologists are committed to this effort.

In eastern philosophy, opportunity and danger are inextricably linked. Despite the threats to medicine in general, anesthesiologists are optimistic about the future and determined to continue the evolution of the specialty. Our practices are more refined, our scientific investigations vigorous, and our ability to impact patient care and costs in the perioperative arena dramatic. Job opportunities are improved¹⁶ and as the population ages, anesthesiology services in the operating room, ICU, pain center, and preoperative clinic will remain in very high demand.

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Primary care and women physicians

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ABSTRACT: *Primary care is on the minds of many politicians, insurance companies, patients, and physicians who have been involved in the tremendous changes occurring in medical care in the United States. The call for more primary care providers has been almost universal, but attracting physicians into the field has been difficult. In the first part of this article, an overview of the field will be presented, followed by an invitation encouraging more women to enter the field.*

Primary care, a supply and demand problem

A few years ago, two medical policy-making groups stated that there will not be enough primary care physicians to meet the future medical needs of the population. In 1992, the Council on Graduate Medical Education (COGME), stated that 50% of graduating medical students should become "generalists" (i.e., enter the fields of family practice, general pediatrics, or general internal medicine).¹ The Association of American Medical Colleges (AAMC) Task Force on Generalist Physicians stated the same.² Currently, only about one-third of medical school graduates choose primary care, and if internal medicine and pediatric subspecialties are excluded, the real number may even be 25% less.³ The U.S. population has grown 43% between 1960 and 1995, and in the same time period, the total number of physicians has quadrupled, leading to almost double the number of physicians per 100 000. With this increase, the number of primary care physicians has remained at 75 to 85 per 100 000. Meanwhile, the need for both specialty and generalist services has increased. As our knowledge and abilities grow along with the need for more preventive services, primary care physicians must do more for patients than was done thirty years ago. In addition, due to managed care, the primary care provider provides the entry point for medical specialty services. Pressure from politicians wishing to contain costs and improve

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access to care has made the impending shortage of primary care physicians even more acute.

The marketplace provides evidence of the current situation. A recent study of physician employment advertisements showed that, in 1990, there were four specialty positions for every generalist position advertised. By 1995, this ratio had dropped to 1.8.⁴ The choice of specialty by physicians is also changing. Following a 10-year dip in interest in primary care, there was a 5.5% increase in medical graduates entering primary care fields in 1996.⁵ However, this will not be nearly enough to adequately meet the medical needs of the population. It is estimated that even if the advised 50% of graduates enter primary care, there will be severe shortages in the near future, and the number of *practicing* primary care physicians would not reach 50% until the year 2040.³

Why did the interest in primary care fall so low? In 1991, only 16% of graduates were seeking primary care careers. For some physicians, the reasons were financial; primary care physicians make less money than specialists. However, another reason may be a negative attitude toward primary care by academic medicine. Educating the primary care physician has not been a high priority in academic centers where research and specialization are promoted. Hunt, et al., showed that 17% of students *changed* their career choice because of negative attitudes expressed by faculty and peers towards primary care.⁶ Colwill stated that "Academic medical centers emphasize the application of science and technology...generalism tends to be defined in terms of the absence of specialization rather than in terms of its positive features of breadth, comprehensiveness, and integration. Most medical education occurs in the tertiary-care milieu, producing a major socializing force toward specialization."⁷ The same author, noted that "medical faculties tend to replicate themselves."⁷ One particularly alarming study found that 30% of the clinical faculty do not see generalists as best suited to provide care for *less serious illnesses*, which "raises questions of what role they do perceive for generalists in the healthcare system."⁸ The study goes on to note "the near universality of the perception that academically capable students are not encouraged toward primary care [which] is another indicator of the low regard for generalism in the academic medicine community."

To solve the problem, the chairman of the AAMC Task Force on Generalist Physicians stated in a recent article "...placing the generalist perspective in the mainstream of undergraduate medical education is the principal goal of the curriculum reforms occurring throughout the country ... [it is] indispensable to the general education of all physicians."⁹ One way to improve the reputation of primary care in medical schools is to increase the number of full-time tenured faculty in this field and, perhaps to, combine family practice, general internal medicine, and general pediatrics into one larger and stronger department. Another solution

may be societal support in the form of free tuition for medical students who choose primary care, as proposed by C. Everett Koop, M.D., former Surgeon General.¹⁰

What kind of physician chooses primary care?

This is a question many medical school deans are asking as they try to attract more generalists. Students who prefer primary care are found to desire patient contact more strongly and to be more interested in serving a diverse population, treating a variety of medical problems, and caring for generally healthy patients.¹¹ In addition, physicians who enter primary care are "people oriented,"¹² and family practitioners have a strong motivation for "direct nurturant patient contact."¹³ Many studies found that those entering primary care were most strongly influenced by personal social values, including a desire to contribute to society.¹⁴ A study using the Schwartz Value Survey found that primary care aspirants rated higher in benevolence values and lower in power and self-direction values than did aspirants to other specialties. It is interesting that women physicians from *all* specialties gave higher ratings to universalism and benevolence values and lower in power than did men.¹⁵

What makes a good primary care physician?

In 1994, the Institute of Medicine added a new term to the definition of primary care, the concept of *sustained partnership* with patients. This would include 1) a whole-person focus, attending to all of the health-related problems of the patient, 2) physician knowledge of the patient, not just of the medical history but of the work, community and cultural context, and the patient's values and ideals about health care, 3) empathy and caring by the physician, 4) patient trust of the physician, 5) appropriately adapted care tailored to reflect the patients' goals and expectations, and 6) patient participation and shared decision making. Antecedents of a sustained partnership include good communication, continuity of care over a long period of time, comprehensive care across all health-related problems, and integration of all health care by the physician regardless of its source.¹⁶ So, a primary care physician must be an adept communicator willing to make a long-term commitment to patients and their families.

Why are women especially suited for primary care?

Women in medicine are increasing at a gradual but continuous rate. In 1995 to 1996, of the 17 357 entering medical school, 7351 (43.2%) were women.¹⁷ Between 1970 and 1995, women physicians increased more than five-fold from 25 401 to 149 404 physicians. In 1995, 68.8% were under the age of 45, in contrast with 48% of their male counterparts.¹⁸ Also of interest, in 1988, only 62% of practicing women physicians were board-certified, as opposed to 82% of their male counterparts. In 1995, 45.9% of women physicians

were in primary care in contrast to 30.3% of men physicians,¹⁹ indicating that women are already inclined to enter primary care. Although generalists make a lower income than specialists, women generalists make 13% more per hour than male generalists.²⁰ Although this is a benefit for women, studies show that for most women physicians, finances are not a deciding factor in choosing their field. This may be because 92% of married women physicians come from dual-income households as opposed to only 45% of their married male colleagues.¹⁸

When a patient comes to a physician, proper diagnosis and treatment is only a part of what is necessary for success; patient understanding and compliance are essential. One study found that building *treatment alliances* were associated with high rates of objectively measured medical compliance over multiple treatment trials.²¹ Another study found a connection between patient satisfaction and compliance and that both were greater for the patients of female physicians.²² Two studies, by Hauck, et al., and Weaver, et al., compared physician *humanistic* behaviors and found a relationship between them and patient satisfaction and compliance.^{23,24}

A common thread in many studies, as well as in the popular literature, is the belief that women are inherently more empathic than men. Empathy refers specifically to the ability of physicians to imagine that they are the patient... an empathic physician imagines what it is like to think, feel, and suffer like the patient.²⁵ "Empathy... provides the patient with a sense of *connectedness* to the physician that may allow him/her to more freely express his/her emotional distress."²⁶

A popular linguistic expert, Deborah Tannen, has written a great deal about the differences in male and female communication techniques. In her book, *You Just Don't Understand*, she presents a strong argument for her belief that "women speak and hear a language of *connection* and intimacy, while men speak and hear a language of status and independence."²⁷ A large study by Debra Roter at Johns Hopkins suggests that female physicians are more empathic than their male colleagues. This and other studies found that female physicians spend significantly more time with their patients.^{28,29} Roter's study found that women physicians talk more to patients than men physicians—about 40% more during history taking. Perhaps more importantly, their patients also talk about 58% more than male physicians' patients. Tannen offers an explanation for this: "Some men really *don't* want to listen at length because they feel it frames them as subordinate. Many women do want to listen, but they expect it to be reciprocal."²⁷

Roter states "The differences in talk evident in male and female physicians' conduct of the history segment of the visit suggests that female physicians may be more attuned to these tasks and more patient-centered in their interviewing

style than men." Barsky has noted that while psychosocial issues are important concerns for patients in primary care, patients' reluctance to voice these problems early in the visit (or at all) creates a 'hidden agenda' of patient concerns.³⁰ Roter concludes "this data suggests there may be less hiding or delay of psychosocial concerns when patients are with female physicians."²⁹ In addition, Ainsworth-Vaughn found in her study that women doctors were much more likely to get the patient's agreement before changing topics, which nurtures a cooperative relationship.³¹ When discussing that many people feel more comfortable having a female explain things to them, Tannen says "If women are focusing on *connections*, they will be motivated to minimize the difference in expertise and to be as comprehensible as possible. Since their goal is to maintain the appearance of similarity and equal status, sharing knowledge helps even the score."²⁷ This may explain why female physicians are more likely than male physicians to review and counsel patients about health behaviors. Several studies have shown that female physicians provide significantly more preventive services than do male physicians, to both female and male patients.^{32,33,34,35,36}

Why do we need more women in primary care?

It has been argued that women should be careful not to fall into the same trap that occurred in Russia where women make up the very large majority of generalists and are "at the bottom of the heap" in that medical system. But, Russia was, until recently, a male-dominated dictatorship. The U.S. system, in contrast, is capitalistic; business and money, more than a central power structure, affect the course of events. The business of medicine in the United States is shifting the power within the medical community irrevocably toward primary care. In contrast to the discouragement received in medical school, primary care physicians discover upon beginning to practice that specialists are dependent on them for referrals. The same group who slighted them are suddenly anxious to communicate, to advise, and to assist with patient care in whatever way they can. There is an intense competition for "kind referrals." This is in sharp contrast to the disrespect experienced in medical school and residency. Now, the primary care physicians' task is to use this shift in power responsibly, ensuring that primary care and specialist physicians forge partnerships to better serve the patients. Women can be especially helpful to this process because, as a group, they are more interested in *connecting* than in maintaining power.

Conclusion

Women, as a group, are better suited to primary care because important parts of it come to us more easily. Not only that, it is a very gratifying field for people who like to

connect to other people. It provides stimulation to those who like to provide comprehensive, integrated health care. It is diagnostically challenging to be the first physician who is seen for most medical problems, and to be the one who coordinates the health-related decisions. It is also stimulating to learn from specialists, and specialists who learn to partner by communicating and coordinating care with primary care providers will be those who receive the most referrals. This is beneficial to the medical care of the patients.

A final reason for encouraging women to enter the primary care field has to do with the changes that are about to occur in academic medicine. The pressure to increase the number of primary care physicians will require many more primary care faculty and new primary care departments. In the past, when there were fewer women entering medicine, and the departments they were entering were solidified and male-dominated, the number of women in the higher tenured faculty positions was historically low.³⁷ The changes which are coming in primary care, with the emphasis on prevention, the importance of partnering with patients and specialists, and increasing communication, satisfaction and compliance, are all areas where a woman's influence and insight are essential. There are wonderful opportunities opening for women in primary care in the fields of research and policy making, which will advance their careers, benefit the field of primary care, and eventually could help to upgrade the status of women in medicine as well.

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What's new in occupational medicine

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ABSTRACT: *Occupational medicine has been a specialty of the American Board of Preventive Medicine since 1955. There are a number of current clinical and administrative topics that are discussed, including employment drug testing, American College of Occupational and Environmental Medicine (ACOEM) guidelines, 24-hour coverage, ergonomics, fiscal accountability, and treating job injuries and workers' compensation claims.*

Introduction

Occupational medicine has been a specialty of the American Board of Preventive Medicine since 1955. In 1950, the World Health Organization (WHO) defined occupational health aims as the promotion and maintenance of the highest degree of physical, mental, and social well-being of workers in all occupations; the prevention among workers of departures from health caused by their working conditions; the protection of workers in their employment from risks resulting from factors adverse to health; the placing and maintenance of the worker in an occupational environment adapted to his physiological and psychological equipment; and to summarize, the adaptation of work to man and of each man to his job.¹

There are many clinical and administrative issues in occupational medicine that deserve comment: drug testing, 24-hour coverage, American College of Occupational and Environmental Medicine (ACOEM) practice guidelines, fiscal accountability, ergonomics, the handling of injuries on the job, and workers compensation claims.

Employment drug testing

Workplace drug testing is not a new concept. In fact, a recent survey by the American Management Association shows that employment drug testing in large businesses has essentially stabilized, but it is still a growing market for small businesses.

Some of the new things in employment drug testing are looking at new mediums for drug analysis. Urine is and has been the main medium for drug testing. It is a proven technology that has been used and tested for decades. The consistency and reliability of urine testing has made it the gold standard for drug testing. But, because of some of its limitations including susceptibility to tampering and ability to detect only short-term use, other mediums are emerging: hair, saliva, and sweat.

Of these three mediums, hair analysis has the largest market size. Hair analysis has many advantages. It provides long-term information and is less subject to evasive maneuvers such as substitutions or specimen tampering. It is an exceptionally stable matrix for entrapped drugs, it does not need special storage or transportation devices, and detection of drugs cannot be avoided by temporary abstention prior to a scheduled test.

However, the weaknesses of hair analysis are important to consider. Hair is a relatively new medium, so not much is known about its characteristics. It is difficult to distinguish between drug use and drug contamination (external). Uptake of drugs is difficult for cosmetically treated hair like perms, coloring, etc. Sex, race, and hair color can potentially bias results, and it is difficult to assess recent drug use because of its relative lag in response time.

In spite of its weaknesses, hair analysis should not be overlooked. Rather than viewing it as competitive to urine, it could be viewed as complementary to urine, with each offering unique potential advantages, while we continue to research hair analysis.

Psychemedics, the only company that has the patent for hair analysis, is based in Massachusetts with its laboratory in California. It provides hair testing services to over 500 businesses. Initial testing is done by radioimmunoassay and confirmatory testing by Gas chromatography/mass spectrometry (GC/MS).

Also new in the drug testing market are several "on-site" drug testing kits. The concept of on-site testing is not new to the workplace, but recently has been gaining more acceptance because of its convenience and immediacy. It can be invaluable to safety-sensitive and time-sensitive businesses. It also can be an important tool for businesses with high employee turnover, remote job sites, or aggressive deadlines. Since on-site testing is not regulated, there are problems both with the technology as well as with work sites that are doing on-site testing. Unless manufacturers of on-site kits and work sites that do on-site testing develop standard operating guidelines, there will continue to be problems with this point-of-care testing.²

ACOEM guidelines

Also new in occupational medicine are the occupational medicine practice guidelines put out by the American College of Occupational and Environmental Medicine (ACOEM).³ These guidelines are an attempt to standardize the evaluation and management of occupational health problems and to reduce

variances in practices. The main target audiences for these guidelines are occupational and primary care practitioners who see workers with occupational health concerns, including illnesses and injuries, and specialists who see presenting problems. It may also prove useful to the specialist to whom occupational medicine practitioners refer concerned, ill, or injured workers, as well as to injured workers themselves, their employers, and insurers of medical care. Secondary audiences include case managers in medicine and managed-care organizations, unions, employee groups, and consumer groups.

The ACOEM practice guidelines are based on presenting symptoms and signs rather than diagnoses, as injured workers enter the medical system with complaints that must first be accurately diagnosed before appropriate treatment, education, and prevention can be provided. Diagnostic consideration from the medical history and physical examination are emphasized. Situations dictating immediate specialty care are reviewed. The guidelines cover initial management, including management of work ability and return to work, special tests, reassessment, and the management of delayed recovery.

The focus of the guidelines is on musculoskeletal and related complaints since they account for over 90% of the total benefits paid for workers compensation, medical care, and temporary disability.

24-hour coverage

Occupational health has changed over the years in its role and responsibility. There has been a transition in the scope of activities. Industrial health programs consisted of small numbers of staff who provided for the immediate needs of employees. These programs provided accident care, pre-employment exams, and assisted employees with medical referrals. Federal, state, and local regulatory agencies forced a change to occupational medicine. In order to meet certain standards and regulations, industries hired in-house staff to ensure compliance. Programs included mandated physical exams, drug testing, surveillance exams, safety, and industrial hygiene activities. A further expansion to occupational health has recently occurred. Employees' health is of utmost interest to the occupational medicine physician. Managing employees who are sick at work or injured on the job has become a cost-effective and efficient means to assure employee productivity for corporations. Wellness or preventive medicine programs are commonplace and have proven to be of value. The final component in this ever expanding field of occupational health is now referred to as corporate health. The overall health of the employee has become the responsibility of the corporate medical director. This is sometimes referred to as 24-hour care and management. All health care of the employee is integrated into one system approach. The occupational medicine physician and staff have responsibility for all the health care of their work force, as well as continuous improvement through outcome measurements.

The following components are included: 24-hour medical coverage; 24-hour disease coverage; 24-hour accident coverage; and 24-hour disability coverage. This systematic approach provides quality care for employees who are productive in the workplace.

Ergonomics

Ergonomics is the study of the interaction of the worker and his environment. The environment affects worker performance, health, and safety in a variety of ways. Every year millions of Americans succumb to the physical and mental stresses created by uncomfortable workstations and poorly designed, low-cost tools and equipment. According to the Bureau of Labor Statistics, the incidence of compensable disorders associated with repetitive trauma disorders in the manufacturing sector alone increased from 4.1 cases per 100 000 workers in 1984 to 29.7 cases per 100 000 workers in 1991. As the cost of health care increases, corporations look to health professionals to assist in reducing their costs related to injuries and workers' compensation. Occupational medicine physicians, ergonomists, industrial hygienists, and other safety and health specialists are finding it useful to work together to evaluate job procedures, equipment, and working conditions. These teams of professionals tour work facilities on a regular basis with emphasis on areas where injuries most frequently occur. This is called worksite/job analysis and is an integral part of any comprehensive ergonomics program because it provides a process for identifying potentially harmful exposures. Problems in job design and activities can be identified and earmarked for possible job restructuring, re-engineering, or job simplification.

Strategies to reduce ergonomic injury should meet several requirements: anthropometric — does the machinery fit the worker?; biomechanical — gripping, lifting, twisting and other movements as part of the job; environmental factors — lighting, climate, space, and noise; psychological needs — job satisfaction and motivation; and personal needs — vary based on experience, training, and physical characteristics of the individual. Computer and epidemiologic models are the latest methods being used to identify hazards and develop solutions. Applying the principles of ergonomics should reduce compensation claims, improve productivity, health, safety, and the psychological well-being of workers.⁴

Fiscal accountability

All areas of medicine require cost management, and occupational medicine is not an exception. In most cases, the occupational health programs are considered support services to the corporate offices. In today's competitive environment, the cost of all these support services is carefully scrutinized. It is imperative that occupational medicine physicians be prepared to apply business standards to their practice. All programs must be able to withstand a critical cost effectiveness and efficiency

test. Outcome data and patient/employee satisfaction divided by the cost of the service will give a better understanding of the services' value. Programs must be benchmarked against other outside programs. Decisions regarding the long-term viability of any occupational medicine program with a corporation rest with fiscal management at all levels of an organization. Answers to any quality or efficiency questions should be answered long before the bottom line scrutiny begins. This entails an educational response for corporate decision makers, and this process demands an understanding of the business of medicine by the occupational medicine professional. Outsourcing may provide opportunities for businesses to save dollars and guarantee compliance with standards and regulations, as well as provide mandated services for employees. It is certain that occupational health services will continue to be provided. The question is where the organization will reside: within the corporate offices, in hospital-based practices, or as free-standing private practices? Understanding the business of medicine and practicing fiscal accountability are special challenges for the occupational medicine physician.

Treating job injuries and workers' compensation claims

Treating injured workers is a major responsibility of occupational health practitioners. This is ideally done at a clinic on the work site by in-house staff.

Prompt, appropriate care is given with close follow-up until full recovery. In-house staff are most familiar with the work environment and the workers. A professional relationship is established with confidence and trust on both sides. Referrals to outside specialists are arranged as needed. Should absence due to work injuries occur, employees can be followed closely to ensure that the best of care is provided and full recovery assured. Causes of injuries can be investigated to prevent similar recurrences. Workers' safety and health are of paramount importance.

Handling workers' compensation in-house is also quite cost effective. All claims can be reviewed by an attorney, administrator, and occupational health physicians on a regular basis. All bills can also be reviewed. This ensures that services rendered were appropriate and fees charged were reasonable. The bills can then be processed promptly. (Cost savings can be up to 20% based on our own experience.)³

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Glaucoma: A preventable cause of blindness

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ABSTRACT: *Glaucoma is a leading cause of blindness within the United States and the leading cause of blindness among African-Americans. Measurement of intraocular pressure only is no longer considered adequate for screening. Recognition of risk factors and examination of the optic nerve are key strategies to identify individuals at risk. Medical and surgical treatment of glaucoma have significantly improved in recent years. Early diagnosis and appropriate therapy will aid in reducing the potential of irreversible blindness.*

Introduction

Ophthalmology is a technologically driven field that has seen significant changes in the last 25 years. Marked progress has been made due to federally supported research and philanthropy. The National Eye Institute of the National Institutes of Health, under the leadership of Dr. Carl Kupfer, has laid the groundwork for enhanced progress in the 21st century. We certainly recognize the progress that has been made with laser therapy of diabetic retinopathy. The risk of severe visual loss due to diabetic retinopathy can be markedly reduced from 50% to 5% with early diagnosis and appropriate treatment. Macular degeneration and glaucoma are two other diseases of the eye that remain the focus of ongoing research. This brief article will focus on glaucoma, a group of eye diseases that is often not diagnosed and can lead to blindness if untreated or undertreated. Moreover, the therapy for glaucoma can significantly influence the quality of life of patients. Thus, early diagnosis is important. Furthermore, significant advances have been made in the medical and surgical treatment of glaucoma.

BIOSKETCH

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Figure 1. An optic nerve of a patient with early glaucoma. Note the notch in the neuroretinal rim at 6:00 indicating focal loss.

What is Glaucoma?

The term glaucoma refers to a group of eye diseases that may or may not be related to an increased pressure within the eye. The aqueous outflow pathway normally drains aqueous as it is produced by the ciliary body. However, as some of us age, the ability of the fluid to drain diminishes to the point that pressure builds within the eye. The optic nerve becomes injured in the process, either through mechanical means related to the pressure at the level of the lamina cribrosa or vascular insufficiency. Over time, the patient will experience loss of peripheral vision without symptoms until the field loss invades central fixation.¹

Most individuals will present with open angle glaucoma. In this country, approximately 20% of individuals will develop angle closure glaucoma. The aqueous outflow pathway referred to earlier may precipitously close in the acute form with resulting marked elevation of intraocular pressure. Patients present with ocular redness, decreased vision, and sometimes nausea and vomiting. Alternatively, the closure of the angle can be more insidious. The patient may present with increased pressure and/or decreased vision on a routine eye exam. With early diagnosis, the acute and the chronic form of angle closure glaucoma can be treated using laser. When an opening is created in the iris, otherwise known as an iridotomy, the pressure between the anterior chamber and posterior chamber equalizes and the iris falls away from the trabecular meshwork, as long as there are no permanent adhesions (peripheral anterior synechiae). If there is permanent closure of the angle, then the patient may

require chronic administration of antiglaucoma medications to control intraocular pressure.

The Prevalence of Glaucoma

Glaucoma remains the third leading cause of preventable blindness in the world and the leading cause of blindness among African-Americans. The prevalence of glaucoma is three to four times greater among African-Americans compared to Caucasians, and the rate of blindness is eight times greater. In the United States, it is estimated that two to three million people have glaucoma and half are not aware. In the state of Maryland, approximately 40 000 individuals over the age of 40 may have glaucoma.

The most important strategy for preventing glaucoma-related blindness is early detection. Comprehensive eye examinations through dilated pupils are required. Once glaucoma is diagnosed, therapy is instituted. As is true with any chronic disease, compliance with therapy is very important. Furthermore, since at least one-third of African-Americans with glaucoma has diabetes and two-thirds have been diagnosed with hypertension, it is important that compliance with treatment for these diseases is maximized as well.

Non-ophthalmologists should be aware of the risk factors for glaucoma and recognize that screening using intraocular pressure is no longer conventional wisdom. Considering that a screener may have only a 50% chance of noting an elevated intraocular pressure in a known glaucoma patient, a careful examination of the optic nerve is a more reliable strategy (See Figure 1). Furthermore, not every patient with an increase in pressure has glaucoma, considering that glaucoma is defined by either an abnormal visual field or optic nerve. Table 1 lists major risk factors for glaucoma. Diabetes and hypertension, once thought to be risk factors, are now recognized as concomitant diseases of the elderly.

Medical and Surgical Therapy of Glaucoma

There are five categories of medications that are used to treat chronic glaucoma: adrenergic antagonists; parasympathomimetics; adrenergic agonists; carbonic anhydrase inhibitors; and prostaglandin analogs. Adrenergic antagonists and carbonic anhydrase inhibitors reduce aqueous production. Parasympathomimetics, adrenergic ago-

nists, and prostaglandin analogs work primarily by increasing the egress of fluid from the eye.

These medications are associated with side effects which can influence quality of life and interact with other prescribed medications. For example, adrenergic antagonists can result in bradycardia, bronchospasm, decreased systolic blood pressure, depression, and memory loss. In combination with systemic medications such as quinidine,² systemic levels of topical beta blocker can be enhanced, thus increasing the likelihood of side effects. Other medications that are prescribed, such as alpha₂ agonists, parasympathomimetics, and carbonic anhydrase inhibitors, can contribute to feelings of malaise. Many patients do not consider topical medications "real" medications and thus may not report their use of eyedrops unless specifically asked. Thus, primary care providers must always ask about eyedrops, particularly if sudden changes in systemic well-being are noted in a given patient. Maneuvers such as punctal occlusion or eyelid closure can significantly reduce the amount of absorbed drug.³

There have been several new advances in medical therapy within the last five years. Recently, a new glaucoma medication, latanoprost, appears to be as effective as beta blockers when given once a day.⁴ This prostaglandin analog has no reported systemic side effects. However, locally in some patients, there is darkening of the color of the iris and growth of eyelashes.

Other new drugs include a topical carbonic anhydrase inhibitor, dorzolamide, which avoids the systemic side effects of the oral agents such as acetazolamide. Alpha₂ agonists are new additions as well. These agents reduce the local side effect of ocular redness and adrenochrome deposits seen more frequently with topical epinephrine.

If medicines are ineffective in lowering intraocular pressure, the patient may need to undergo either incisional surgery or laser trabeculoplasty, a noninvasive technique which involves treating the aqueous outflow pathway with either argon or diode laser. Incisional surgery is invasive; a small opening is created in the trabecular meshwork and the anterior chamber is linked to the subconjunctival space.

There have been a few advances in surgical therapy as well in recent years. Antimetabolites, such as five fluorouracil and mitomycin-C, when used in filtration surgery, have enhanced the surgical success for many patients who, in the

past, failed this procedure.⁵ Moreover, new laser delivery systems, such as the diode lasers, have provided the opportunity to replace a painful procedure, cyclocryotherapy, with a laser procedure that is as effective and easier for patients who are refractory to other forms of therapy. Finally, glaucoma drainage devices⁶ can also be used in eyes which are considered high risk for failure following conventional filtration surgery.

Summary

Glaucoma will result in blindness if untreated or undertreated. Patients with significant risk factors or a suspicious optic nerve examination should be referred for further evaluation. Patients who are diagnosed need to be encouraged to continue their medication and see their ophthalmologist regularly. There are a number of options available to patients now that were not available a decade ago. However, patients at risk must be identified at a stage early enough to avoid extensive optic nerve damage and visual field loss. It will take the entire community to eradicate this preventable cause of blindness.

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Table 1. Risk Factors for Glaucoma

- ▼ Age
- ▼ Race*
- ▼ Family history
- ▼ Trauma
- ▼ Chronic use of steroids
- ▼ Last comprehensive eye exam >2 years

*Patients who are of African descent

Glaucoma 2001 is a public service project of the American Academy of Ophthalmology. Patients with significant risk factors, regardless of insurance, may be referred for a comprehensive eye examination. Please call 1-800-391-3937 for more information.

Hormonal strategies of the menopause

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ABSTRACT: A woman at the age of 50 in the United States has a life expectancy of approximately 30 years. The menopausal years, the symptoms associated with menopause, and concurrent conditions associated with these age groups are of major importance, since women may live up to one third of their lives in menopause. In this discussion, the physiologic conditions associated with menopause, including osteoporosis, cardiovascular disease, and breast cancer, are considered, with specific assessment of risks and benefits of hormone replacement therapy. Specific hormone replacement regimens are reviewed, along with non-estrogenic approaches to osteoporosis therapy. The long-term benefits of estrogen on female life expectancy are reviewed, balancing cardiovascular and osteoporosis benefits within the context of long-term effects on breast cancer risk.

BIOSKETCH

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Female life expectancy in the United States has changed significantly since the turn of the century. In 1900, the age of menopause was approximately 50 years of age, with life expectancy of 59 years. By the year 2000, female life expectancy is projected at 78 years, with menopause occurring at the mean age of 51.4 years.¹ This represents almost a 30-year segment of women's lives with special concerns and a need for comprehensive preventative health care. The world population of women age 45 and over is increasing; with approximately 251 million women in this age group projected by the year 2000 in developed countries, and 468 million in developing countries, a total of 719 million women age 45 and over are projected in the world by the year 2000.^{2,3} This represents a significant segment of the popula-

tion for the millennium. The major health concerns facing women age 45 and older are menopausal symptomatology, osteoporosis, cardiac disease, and breast cancer.

Menopausal symptomatology and decision making regarding estrogen replacement therapy

The symptom complex facing most menopausal women includes hot flashes,^{4,5} alteration of menses, vaginal atrophy, and alterations in libido.⁶ Hormone replacement therapy (HRT) alleviates hot flashes early on in most women.⁷ In addition, prevention and relief of urethral, bladder,⁸ and vaginal atrophy⁹ is accomplished with HRT as well as maintenance of collagen levels in the dermis.¹⁰ Less depression and improvement in Alzheimer's disease symptomatology has been reported in recent studies of women on HRT.¹¹

Commonly utilized estrogen and progestogen prescribing schedules include sequential therapy or continuous daily therapy.¹² Estrogens and progestogen are usually prescribed in combination in women with intact uteri. The incidence of vaginal bleeding varies with the type of HRT regimen prescribed. The sequential regimen consists of estrogens administered from days 1 to 25, plus medroxyprogesterone acetate 10 mg administered from days 16 to 25 or medroxyprogesterone acetate 5 mg administered from day 13 to 25. The patient will usually experience predictable menses from days 26 to 30.¹³ It is often useful to begin sequential HRT on the first of the calendar month. The continuous regimen consists of estrogen daily plus medroxyprogesterone acetate 2.5 mg daily.¹⁴ Usually during the initiation of continuous regimen, there is an increased incidence of irregular vaginal staining which appears to decline within three to six months, so that no menses ultimately occur.¹⁵ Oral estrogen doses range from 0.625 mg to 1.25 mg of conjugated equine estrogens, 1.0 mg to 2.0 mg of micronized estradiol,¹⁶ 0.625 mg to 1.25 mg of estrone sulfate, or 0.05 mg to 0.10 mg of estradiol administered through a weekly transdermal patch.¹⁷ Adjustments in medication may be necessary, particularly during the perimenopausal period; however, it is recommended to maintain dosage at the equivalent of .625 mg of conjugated equine estrogens for long-term maintenance. Recently, a new vaginal estradiol ring inserted for 90 days has been approved for use in menopausal therapy. Topical estrogen therapy is often utilized for genital atrophy.

Osteoporosis

Postmenopausal osteoporosis affects over 20 million American women, with 1.3 million osteoporosis fractures occurring annually. One out of three women will suffer a vertebral fracture after age 85, and one out of three women will suffer a hip fracture after 85. Most women will have a one-in-three chance of developing an osteoporosis-related fracture in their lifetime, and approximately 65 000 women die annually from hip-related fractures.^{18,19} The most rapid decline in bone mineral density occurs in the first ten years after menopause, and by age 85, more than 30% of the mass at age 50 may be lost.²⁰ Bone densitometry monitoring, specifically dual energy x-ray absorptiometry (DEXA) scanning or dual photon absorptiometry^{21,22} is useful in the evaluation of the current status of trabecular bone in the menopausal woman.

After initiation of HRT, the rapid decline in bone density occurring during menopause is slowed, with a decreased risk of fractures of the hip, spine, and forearm in most women.^{23,23} If HRT is started well into the menopause, the bone mass already lost will not be made up, but a decline in significant loss should occur.²⁵ Attention to exercise, nutrition, and other medications that may impact bone density is of importance. Calcium intake is mandatory, and 1500 mg of calcium per day is recommended as a dietary supplement in women who have little or no dairy intake in their diet.²⁶ Currently available calcium preparations include oyster shell calcium carbonate 500 mg tablets or over the counter antacids such as Tums, which are calcium carbonate based. Calcium, however, is not sufficient treatment alone in the postmenopausal period and should be combined with HRT if possible.

New alternatives for the treatment of osteoporosis include alendronate, an osteoclast inhibitor which was recently FDA approved for the treatment of postmenopausal osteoporosis. Increases in bone mineral density of up to 10% have been reported after the administration of alendronate for a period of three years.²⁷ This medication is administered in a 10 mg dose in the morning on an empty stomach with no oral ingestion for at least 30 minutes. Other medications useful for the prevention of osteoporosis include calcitonin, which is available as an injectable and nasal spray preparation.²⁸ Doses of the injectable calcitonin range from 100 units daily to 50 units three times a week. Nasal spray calcitonin, recently approved, is dosed in 200 units (one spray) per day. In addition, modification of lifestyle factors, such as cessa-

tion of smoking and limitation of alcohol and caffeine, is also important in the prevention of osteoporosis.²⁸

Cardiac disease

Cardiac disease is a significant health risk in American women. One-in-two postmenopausal women will develop heart disease in the United States,³⁰ and coronary artery disease kills approximately 233 000 women annually.^{31,32} Among American women, more than 50% have at least one risk factor for coronary heart disease including hypertension, elevated serum cholesterol levels, and smoking.^{33,34} HRT has been shown to significantly decrease the risk of coronary heart disease in the female and actually results in a gain in life expectancy in women at significant risk for heart disease.^{35,36,37} HRT has been reported to decrease coronary occlusion scores.³⁸ A markedly decreased relative risk of myocardial infarction has been demonstrated in women on the medication, including women with multiple cardiac risk factors.³⁹ Estrogen appears to decrease the binding of LDL cholesterol to vessel walls, as well as increasing HDL cholesterol and decreasing LDL cholesterol values, which results in a favorable cardiac lipid profile.⁴⁰

Breast cancer

Breast cancer kills approximately 43 000 women yearly in the United States³¹ and women have a one-in-eight lifetime chance of developing breast cancer.⁴¹ It is important to consider breast cancer evaluation in a menopausal patient with annual mammography and careful breast evaluation. In addition, the benefit of HRT balanced with the risk of breast cancer needs to be evaluated in the context of each individual patient. In recent studies, HRT was shown to have a slightly increased relative risk of breast cancer when used for over five to ten years.^{42,43} Subsequent studies have also shown that a group that would not benefit from HRT would be those with two first degree relatives with breast cancer, a group of approximately 1% to 2% of the population of women in the United States.^{44,45}

In conclusion, most women entering menopause, a segment of their lives which could encompass 30 years, would benefit from HRT. An overall increase in life expectancy may occur due to significant improvement in the incidence of osteoporosis-related fractures and a dramatic decrease in cardiac risk, while balancing the risk of breast cancer. Current investigations of new pharmaco-

logic agents are also promising with respect to non-estrogenic approaches to decreasing osteoporosis and cardiovascular disease in menopausal women.

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The role of large core needle biopsy in locally advanced breast cancer

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ABSTRACT: *The appropriate management of locally advanced breast carcinoma (LABC) is controversial. It is evident that overall survival in patients treated with locoregional control remains dismal. The trend towards a more effective means of improving response rates and overall survival in LABC has shifted towards earlier aggressive treatment to include neoadjuvant chemotherapy. A known histopathological diagnosis is required prior to the implementation of therapy, which is dependent upon the initial method used to obtain tissue. We advocate the use of large core (14g) needle breast biopsy over other commonly employed techniques. This technique is reliable, safe, and efficient.*

Introduction

The American Cancer Society estimates that, in 1996, 184 300 women will be diagnosed with breast cancer in the United States and 44 300 will die from this disease.¹ Despite rising breast cancer awareness through public education and screening programs, the incidence of patients presenting with locally advanced disease approaches 10% to 20%.²⁻⁴ Size of the primary tumor and nodal involvement are the two most important prognostic factors, with ten-year survival ranging from 82% for stage I disease (primary under 2 cm, node negative) to 37% for stage III disease (primary over 5 cm, with ipsilateral axillary metastases).⁵ Treatment of stage III breast cancer traditionally involves mastectomy followed by chemotherapy and radiation therapy. Recent reports describe the use of neoadjuvant chemotherapy to reduce tumor burden prior to surgery.⁶⁻¹⁰ In a series of 165 women planning mastectomy for tumors larger than 3 cm in diameter (range 3 cm to >7 cm, median 4.1 cm to 5 cm), Bonnadonna et al., observed tumor shrinkage to less than 3 cm in 127 (81%) following

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neoadjuvant chemotherapy, allowing breast conservation to be performed.⁶ Complete histopathologic remission was documented in seven patients. We advocate the use of large core (14g) needle breast biopsy for initial diagnosis in evaluating patients under consideration for neoadjuvant chemotherapy and present a discussion of the advantages of core needle biopsy over other means of tissue diagnosis.

Materials and methods

From July 1995 through June 1997, at the University of Maryland Medical Center, a total of 383 core needle breast biopsies were performed using either ultrasound (n = 236) or stereotactic (n = 147) guidance; 104 (26%) proved malignant, with four of these initially diagnosed as atypical ductal hyperplasia. Eighty-four lesions proved to be stage 0-II disease; 80 of these were surgically excised. Fourteen lesions were seen in stage 0 disease, 28 stage I, and 36 stage II. One proved to be a mucosal-associated lymphoid tumor and is awaiting surgery. Surgery is pending for three other patients, and in two, the surgical outcome is unknown. Twenty core biopsies were performed (in 16 patients, 15 women and 1 man) for tissue diagnosis prior to initiation of chemotherapy for advanced disease and form the basis of discussion. Average patient age was 61.4 years (range 40 to 94) and lesion size 4.3 cm (range 1.0 cm to >15 cm). A 14g Monopty gun (Bard, Covington, Georgia) was employed for all biopsies. Deliberate sampling from different areas of each mass was performed, with needle position documented by ultrasound (10MHz linear array transducer, Acoustic Imaging Performa, Phoenix, Arizona). An average of 5.2 passes (range 4 to 6) were made through each lesion. In three lesions, moderate bleeding was noted which resolved after five minutes of manual compression with no evidence of hematoma sonographically. No other complications were noted. In three patients, the diagnosis of lymphoma was suspected; a fourth had a history of chronic myelogenous leukemia. In these cases, tissue samples were placed on saline-soaked gauze (rather than in formalin) to allow for flow cytometry, cytogenetic, and molecular studies.

Results

Histopathologic diagnoses were: six infiltrating ductal carcinoma (four of which had a small component of DCIS); three infiltrating lobular carcinoma; five poorly differentiated adenocarcinomas (two considered primary breast cancer; one local chest wall recurrence of breast cancer; and two considered metastatic from opposite breast cancer); two metastatic lymphoma; two metastatic lung cancer; one chlo-

roma; and one lymph node metastasis from breast cancer (**Figure 1**). All patients underwent chemotherapy on the basis of the core needle biopsy results. Nine patients were determined to have stage IV disease and were treated with chemotherapy alone; one of these patients died within two weeks of the core needle biopsy, presumably related to extensive lung and liver metastases. Two patients with stage III disease were treated with neoadjuvant chemotherapy with reduction in the tumor size; in one patient, tumor decreased from 35 mm to 20 mm and breast-conserving surgery was performed. The second patient had mastectomy after the tumor decreased from 50 mm to 20 mm in greatest diameter. The two patients with metastatic lung cancer, the two with lymphoma, and the one with a chloroma underwent chemotherapy on the basis of the core needle breast biopsy results.

Discussion

For clinically and/or mammographically suspicious breast masses, tissue diagnosis can be achieved primarily by three methods: fine needle aspiration (FNA), large core needle biopsy, and excisional (or incisional) surgical biopsy. In contrast to FNA, large core (14g) needle biopsy yields sufficient tissue for detailed histopathologic diagnosis without the morbidity associated with open surgical biopsy. Overall sensitivity of FNA ranges from 68% to 93%, with specificity 88% to 100%.^{11,12} Insufficiency rates for FNA range from 0% to 38%,¹¹ with 9% of such cases proving malignant across multiple series.¹³ Another 3% to 25 % of FNAs are considered atypical, of which 0% to 49% prove to be malignant.^{14,15}

For 14g core needle biopsy, overall sensitivity has been reported at 85% to 97%, with no false positive findings of malignancy.¹⁶⁻²¹ An 8% to 12% underdiagnosis rate has been observed for core needle biopsy across several series.^{18, 22-25} In these cases, the material sampled may not be fully representative; an infiltrating ductal carcinoma may be a minor component of a lesion in which only intraductal carcinoma has been sampled. In several series, from 19% to 20% of cases determined to be *in situ* on the basis of core needle biopsy will prove also to have an infiltrating component at excision.^{22, 26} Another source of underdiagnosis is difficulty distinguishing atypical ductal hyperplasia from intraductal or infiltrating cribriform ductal carcinoma; from 52% to 56% of cases diagnosed as atypical ductal hyperplasia on 14g core biopsy will prove malignant at excision.^{22, 23} With the development of vacuum-assisted 14g and 11g probes (Biopsys Medical, Irvine, California), two- to five-fold greater

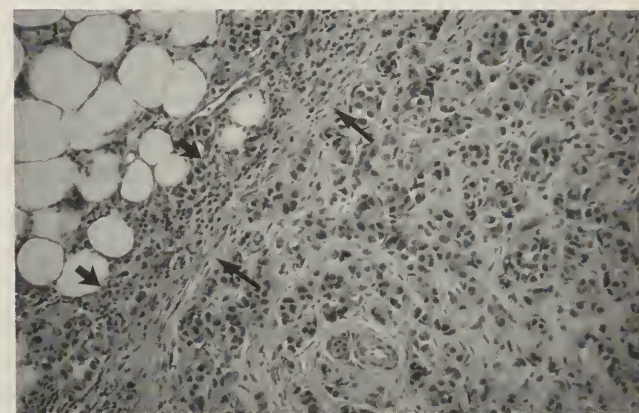
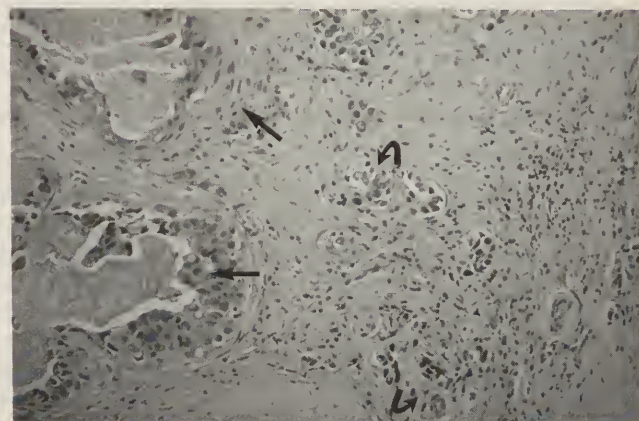
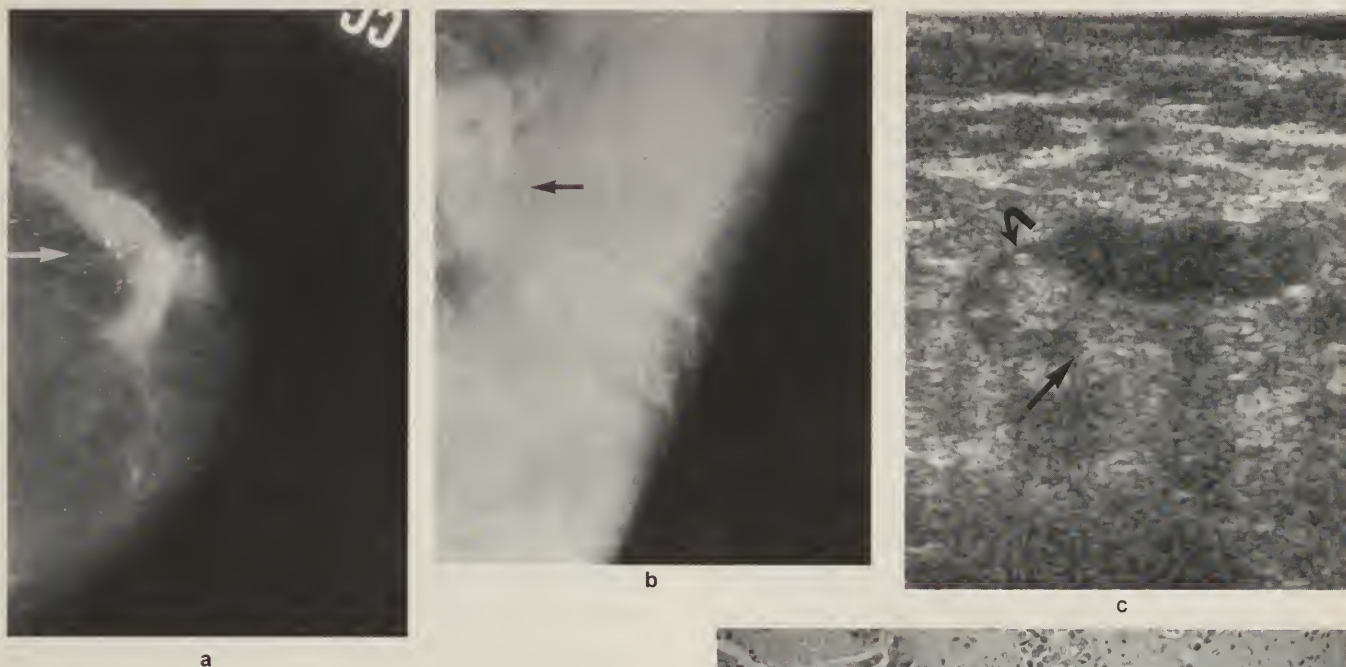


Figure 1. 56-year-old woman with stage III breast carcinoma, confirmed on core needle biopsy of primary tumor and lymph node metastasis. a) Craniocaudal mammogram demonstrates a dense 50mm spiculated mass with associated linear branching microcalcifications (arrow) in the upper outer right breast, proven to be infiltrating and intraductal carcinoma, involving the nipple. A small, 3 mm satellite focus of pleomorphic calcifications was evident more posteriorly on the mediolateral oblique mammogram. The opposite breast appeared normal. b) Magnification mammographic view of the right axilla demonstrates microcalcifications superimposed on at least one enlarged lymph node, proven to be metastatic breast carcinoma. c) Sagittal sonographic image of the axillary node again demonstrates the calcifications (curved black arrow) and revealed focal bulging of the contour, though the central echogenic fatty hilus was retained. d) Histopathology of the 14g core needle biopsy of the primary tumor shows two foci of intraductal carcinoma (black arrows) with nests of tumor cells invading the stroma (curved black arrow), H&E, 40X. e) Histopathology of core needle biopsy through the suspicious node shows metastatic infiltrating ductal carcinoma (short black arrows) with tumor cells penetrating beyond the lymph node capsule (black arrows) into the peri-neural fat. The Bloom-Richardson grade was 2/3 with receptors in both the primary and lymph node metastasis positive for estrogen, progesterone, and Ki-67. The patient was referred for treatment of a stage IIIB carcinoma with neoadjuvant chemotherapy consisting of three courses of adriamycin prior to surgery. At mastectomy, the tumor had decreased to 20mm in size. Histopathologic findings at excision confirmed the results at core biopsy.

yields of material are obtained,²⁷ reducing the rate of underestimation of disease to nearly zero.^{28,29} When underdiagnosis has occurred, the patient would still undergo a surgical biopsy. The patient has had an extra procedure but without risk of misdiagnosis. By core biopsy, outright miss of the lesion being sampled has been seen in from 1.1% to 2.5% of masses after five passes;^{21,24,30} this is comparable to the miss

rate of 3% observed for needle localization/excisional biopsy.³¹ With palpable masses, the miss rate would be expected to be essentially zero with either open surgical biopsy or core needle biopsy.

Detailed histopathologic analysis is critical in planning therapy. FNAs cannot reliably differentiate infiltrating from *in situ* carcinoma.^{32,33} While estrogen and progesterone

receptor status can be discerned, Bloom-Richardson grade³⁴ cannot be accurately assessed. Prior to the advent of core needle biopsy, incisional biopsy was required to obtain such information. With locally advanced breast cancer, local healing becomes problematic with incisional biopsy. Further, surgical biopsy carries an increased anesthesia risk compared to local anesthesia (1% lidocaine) used for core biopsy.

The optimal method for obtaining tissue diagnosis in patients with highly suspicious breast masses has been debated. In the past, an initial diagnostic surgical biopsy would typically be performed, followed by definitive surgery. Liberman et al., showed that 77% of women with highly suspicious breast masses were spared at least one surgical procedure by initial performance of a core needle biopsy. Only a single therapeutic surgical procedure was needed in most cases.³⁵ By contrast, most surgeons will not perform axillary node dissection on the basis of FNA results alone, as the infiltrating nature of the tumor usually cannot be established by FNA. Initial diagnosis with core biopsy, in addition to guiding the decision to perform axillary nodal dissection, facilitates obtaining clean margins at the initial surgery. Indeed, in one series, surgery resulted in positive margins in 40/107 (37%) of cancers undergoing initial diagnostic surgical biopsy compared to only 5/62 (8%) of cancers revealed by initial core biopsy.³⁶

The term locally advanced breast carcinoma (LABC) refers to stage III breast carcinoma, as defined by the American Joint Committee (AJC). Stage IIIa represents tumor greater than 5 cm with metastases to the ipsilateral axillary nodes. Stage IIIb represents any size tumor with direct extension to the chest wall or skin, skin edema or ulceration, and inflammatory carcinoma. Although the prognosis is generally poor, combined mastectomy and radiation therapy can improve local control with a five-year survival rate that approaches 38% to 45%.^{37,38} However, it is likely that most of these patients will ultimately develop distant metastases. Therefore, a combined modality approach to include neoadjuvant chemotherapy prior to locoregional therapy is being increasingly utilized in the treatment of patients with LABC when reduction of tumor bulk is the goal prior to surgery.

Combination therapy that includes neoadjuvant chemotherapy in patients with primary lymph node positive stage III breast cancer appears to improve overall survival in various series, ranging from 40% to 65% at five years.^{8,9,39} Several reports of combined modality treatment, which include induction chemotherapy followed by definitive local treatment and adjuvant chemotherapy, demonstrate com-

plete response rates varying from 58% to 100% and three-year disease-free survival ranging from 35% to 78%.⁴⁰ Neoadjuvant chemotherapy is advocated for large but operable breast cancer where the prognosis, even after mastectomy, remains poor. Prognosis is primarily a function of tumor size and lymph node status. The goal of treatment with preoperative chemotherapy is to reduce tumor size and total tumor cell burden, and thereby prolong survival and downstage the primary tumor. This, in turn, allows surgical resection of tumors that were previously considered inoperable.⁴¹ Schwartz et al., report that 10% of patients with LABC had no residual tumor in the breast at surgery. An additional 7% had only duct carcinoma *in situ* and no evidence of invasive carcinoma.⁸ Neoadjuvant chemotherapy also enables assessment of tumor response to a particular drug regimen and consequently, provides a chance to optimize treatment and to improve the cure rate of postoperative (adjuvant) therapy.^{7,8}

In addition to staging and assessment of prognostic factors in patients with locally advanced breast cancer prior to chemotherapy, core needle biopsy affords a specific diagnosis when other malignancies are the source of the breast mass. Two of the patients in our series were shown to have lymphoma. Two other patients in our series were suspected of having breast cancer, but analysis of the core biopsy material allowed the proper diagnosis of metastatic lung cancer to be demonstrated and confirmed by further imaging studies. These patients were spared an unnecessary surgical procedure.

In summary, large core 14g needle biopsy has several advantages over FNA and surgical excision in obtaining initial tissue diagnosis for both a primary breast mass and suspicious lymph nodes. This method facilitates histopathological staging prior to implementing therapy. Overall sensitivity and specificity of diagnosis are improved with core needle biopsy over FNA, and evaluation of histochemical and histopathologic prognostic markers is as effective as with material obtained at surgery. Patients with pathologically proven LABC can then be initially treated with neoadjuvant chemotherapy with reliable knowledge prior to locoregional management.

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Managed care: Physician-based ownership issues

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ABSTRACT: *The late 1990s offers an opportunity for physicians to have equity in a profitable managed care organization. Managed care today is money management to improve the profit for corporations. Quality is desired but not demanded by payors at this time. To compete effectively, physician-owned managed care organizations must be able to demonstrate to the payors their results, showing quality medical care and cost containment.*

Paul M. Ellwood, Jr., M.D., who masterminded the rise of health maintenance organizations (HMOs), says, "The only way doctors will regain control of health care is by running the system themselves. But to do that, they will have to accept managed care. I don't think the game is lost forever. But it's got to be played by a different set of quality-based rules." This is the time for physicians to organize and be able to deliver quality health care with cost containment. Physicians can form or join organizations such as independent practice associations (IPAs), provider sponsored organizations (PSOs), physician hospital organizations (PHOs), managed care organizations (MCOs), and management service organizations (MSOs).

Physicians can accomplish managed care in several ways: 1) Organized physician groups can get the lion's share of HMO premiums by taking responsibility for the cost of medical care through capitation under full risk and full professional risk contracts. HMOs are starting to delegate much of the utilization and quality control that they would otherwise do. A consultant at the actuarial firm Tower Perrin in Overland, Kansas thinks some HMOs would rather get guaranteed portions of the premium than an uncertain shot at a profit. 2) Physician groups in the form of IPAs, PSOs, PHOs, MCOs, or MSOs can contract directly with

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Medicare, Medicaid, business coalitions, or other payors, provided they have a license. Employers can contract directly with providers as a self-insured entity using the Employment Retirement Insurance Security Act (ERISA) plan. For physician groups to have a better chance at getting contracts from payors, they must be able to show data on patient satisfaction, access, cost containment, and clinical and functional status outcomes. Physician groups must have enough capital reserve to pay for good management and reliable sophisticated information systems.

Antitrust

Physician networks must abide with antitrust laws. The law is designed to weed out collusive networks. Some lawyers say that antitrust problems could be avoided if physicians share financial risk through capitation or substantial withholds. The Federal Trade Commission (FTC) and the Department of Justice now say they will consider clinical integration alone as a justification of price setting, even if the doctors do not take monetary risk or integrate their practice financially. Donna D. Fraiche, a New Orleans attorney, defines IPAs as clinically integrated if they have the following characteristics:¹

- ▶ Systems to establish quality and utilization goals
- ▶ Evaluation of individuals' and network's performance as they relate to these goals
- ▶ Case management, some requirements for pre-authorization, and review of inpatient stays
- ▶ Investment in an information system
- ▶ Comparison of network performance with cost and quality benchmarks
- ▶ Monitoring of patient satisfaction
- ▶ Detailing service reports provided to a payer
- ▶ Presence of a medical director and support staff
- ▶ Physician involvement in development of standards and protocols
- ▶ Retention of an agent to negotiate contract fees for non-network patients
- ▶ No physician agreement on fees for patients outside network contracts
- ▶ Physicians permitted to practice outside the IPA
- ▶ The number of IPA physicians in any one specialty constitutes no more than 35% of the market's total number of physicians in that specialty

Medicare risk contracts

The Health Care Financing Administration (HCFA) contracts only with state license entities. However, legislation will be reintroduced this year allowing HCFA to deal directly with provider networks, whether they are state-licensed or not. If and when the legislation is passed, the window of opportunity may not be open long. Physicians have only a few years to seize the entrepreneurial opportunity in Medicare risk contracting. To obtain Medicare risk contracts, there will be bidding processes. Cost and quality issues will be important.

Currently, Medicare reimbursement to HMOs is based on 95% of the "adjusted average *per capita* cost" (AAPCC) that HCFA posts yearly for each county. AAPCCs range from \$300 per member per month (PMPM) to \$600 PMPM in major markets. AAPCC rate information is available from HCFA on the Internet at <http://www.hcfa.gov> under "stats and data."

A MSO can be formed by a provider sponsored network to do all of its management services while reducing the provider's risk. It is better for the MSO to hold the contract to insulate the medical practice in case things do not work out. An MSO can be the vehicle for equity investments by providers and outside investors.

Start-up capital for building infrastructure and hiring seasoned administrators can run into the millions of dollars, and you need enough cash to keep operating until you break even. For them to be successful, IPAs or medical groups need capital, business expertise, administration, and information systems. The physicians must have an incentive on a continuing basis, for example, options for stock ownership and monetary compensation for achieving quality medical care with cost savings.

Solo practitioner and medical groups

A solo practitioner can be a member or an investor in any medical organization such as an IPA, PSO, PHO, or MSO. The physician must be knowledgeable in the dynamic changes of health care in order to make a wise decision on important issues like:

- ▶ How to improve your practice;
- ▶ How to choose which medical organization to join or invest in;
- ▶ Financial and medical legal risks involved;
- ▶ Return on investment; and
- ▶ Financial and medical autonomy.

In some parts of the country, provider groups represented by physicians and hospitals in the form of an IPA, PSO, MSO, or PHO are beginning to eliminate the managed care middleman by contracting directly with employers who pay the bills. They are becoming more organized. They internalize cost control functions. They implement efficient management and software technology in order to achieve their goals. The goal is to get contracts from employers or payors. Groups who can demonstrate to payors cost containment and quality medical care, substantiated by clinical and functional outcome data will get the market.

Practices with global capitation arrangements have a strong incentive to keep their patients healthy and out of hospitals and emergency rooms. Groups that do Medicare risk contracting get most of the Medicare premium in return for providing all the care.

Successful medical groups invest in the infrastructure that enables them to deliver such care, which should increase the profitability of the whole group. For example:

- ▶ Hire case workers and discharge planners to help physi-

cians in keeping their patients healthy and out of the hospital.

- ▶ Assign specific physicians that belong to the group to handle hospital care only. The expertise of the Hospitalist should decrease hospital days.
- ▶ Assign certain physicians to round on all nursing home patients.
- ▶ Create teams for specific diseases; these teams generally consists of internists, specialists, nurse practitioners, and case managers.

Pulmonary rehabilitation centers can be set up in different strategic locations to keep patients functional and away from the emergency room and intensive care units by preventing and correcting in the rehabilitation centers the early phases of congestive heart failure (CHF), bronchitis, wheezing, and pneumonia.

Issues to consider when ownership is offered

Consult your lawyer regarding the proposed contract or initial offering. Find out your position, stock options, obligations, benefits, and restrictions in the managed care organization. Know your entry level of engagement and exit strategies or options. Inquire about the structural organization of the company, who are the principal owners, what is the percentage of ownership, how many existing stocks, and how much and where will funding come from.

An ideal organization to join is one that:

- ▶ Will give incentives to the physicians groups to be creative in keeping their patients healthy, improve quality of life, and practice good medicine with cost containment;
- ▶ Provides stock options and equity;
- ▶ Is capable of providing a sophisticated software technology and can customize off-shelf products to serve the needs of the medical practice to improve health care, contain cost, increase revenue, reduce malpractice risk, collect a data base, push technology, and market capabilities;
- ▶ Has physician ownership as part of the holding company; and
- ▶ Has a structure to provide continuous incentives for both present and future members of the group without violating Stark and Antitrust Laws.

Integrated information systems solution

A sophisticated and reliable information system will do efficient billing, provide financial information, scheduling, and outcome data. Allow consultants and team members access to the patient records with security components so that sensitive medical and financial information can not be accessed. Effective case management and cost containment will result if the team members, consultants, and primary physicians have access to the patient records regarding medical history, allergies, medication, results of lab, x-ray and other diagnostic studies, so that an intelligent discussion and plan imple-

mentation can be carried out without duplication of tests, and problems can be avoided.

Beware of computer software companies that make promises, but cannot deliver. Currently, there is no single vendor solution to do all. The key is to use off-shelf products, realizing they need to be customized.

Helpful suggestions in the choice of your network's software:

- ▶ Open systems compliant with industry standards (i.e., non-proprietary).
- ▶ Scaleable and modular, so it can grow and build on modules as you need more processing power.
- ▶ Maintainable at reasonable cost.
- ▶ System flexible to accommodate change.
- ▶ User-friendly (windows-based.)
- ▶ Network enabled with Internet connectivity and Intranet capability.
- ▶ Browser technology.
- ▶ Robust database environment.
- ▶ Standard-based (e.g. sequel compliant).
- ▶ "Data Warehouse" or a smaller version "Data Mart."

Benefits

It is physicians who control nearly 80% of health care related expenditures in the United States. It is, therefore, physicians who must regain control over the practice of medicine and delivery of patient care. The benefits of physician owned and incentivized organizations are extensive and will be both pervasive and nontransient. Summarized, these benefits include:

- ▶ Better patient care at market driven prices
- ▶ Better quality of life for physicians and other health care providers
- ▶ Reduced fraud, waste, and cost to society of health care related services.

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Review of universal health insurance: Should we try it?

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ABSTRACT: *Health care delivery and finances are fragmented in the United States resulting in inaccessible services to many in need. Other countries with universal health insurance have developed systems that provide basic care for all individuals while maintaining costs in a reasonable range. It is time to make tough decisions to enhance the public's health status and gain control over the destiny of medical practice, while considering the bottom line.*

Delivery of care to U.S. workers

Approximately 75% to 85% of the uninsured are employed or are dependents of workers.^{1,2,3} On April 27, 1996, the *Baltimore Sun* reported that the number of uninsured Americans has increased to 42 million. When employees lose health insurance coverage, they often do not qualify for public assistance, even though they are considered poor due to low wages. Forty-eight percent of the working poor are without health insurance and the reason given is that the insurance is too costly.³

The 1993 Behavior Risk Factor Surveillance System (BRFSS) survey included 81 794 persons aged 18 to 64 who responded to health care coverage questions. Sixteen percent reported being uninsured at the time of the interview (the range was 7% in Hawaii to 26% in Louisiana). The uninsured received less preventive health services and were more likely to be younger, less educated, unemployed, of low income, and of races other than white compared to the insured.⁴ A similar report by Franks,⁵ using the National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (NHEFS) data, showed that the uninsured risked 25% higher mortality rates compared to the insured after adjusting for multiple socio-ecodemographic factors (odds ratio 1.25 [95% confidence interval of 1.00 to 1.55]). The results

suggest increased mortality because of lower quality of care and/or decreased access to health care services.

According to data reported by the Democratic Policy Committee in 1994,⁶ the group aged 25 to 34 is the largest uninsured (23.4%), followed by children under 18 years old (22.1%). The U.S. Census Bureau reported that young adults aged 18 to 24 were more likely to lack coverage during 1995 than other groups.⁷ About 72% of the uninsured Americans have incomes above poverty. Forty-eight percent of the uninsured workers are self-employed or work in firms with fewer than 25 employees. Of those workers not covered by employer-paid insurance, some are eligible for workers' compensation or union insurance, many pay out of pocket, attempt to obtain coverage by public assistance, or do without coverage. This latter group still may seek care in the event of illness or injury, but the cost of the care is then assumed by the taxpayers as uncompensated care.

Approximately 70% of small businesses insure their employees, and in the fall of 1996, the Health Insurance Portability and Accountability Act was enacted to further protect workers who change jobs or are faced with serious illnesses.⁸ COBRA (Consolidated Omnibus Budget Reconciliation Act) also allows employees to maintain the insurance they held under previous employers for 18 months after leaving. The drawback is employees are responsible for the total premium costs, which are usually very expensive.

Workers' compensation covers medical treatments for job-related injuries and also makes payments to workers who are away from work due to those injuries.⁹ Some industries cover their workers through union health and welfare trust funds, which offer the typical benefits that group health plans do, such as hospitalization, major medical, and prescription drugs. They rarely offer occupational health or preventive services.¹⁰ Some are beginning to provide preventive services in a managed care setting, usually offering only a basic annual physical exam.

The major problem businesses face in trying to provide health insurance coverage to their employees is the excessive costs, especially with small firms. In 1995, the average cost per employee (total health benefit cost) was \$3,821 for annual health insurance.¹¹ As a result of increases in the cost of providing medical coverage for employees, some employers have refused employee pay raises, reduced wages, added costs onto their goods so the consumer helps pay, and in some cases, they have eliminated health care coverage or reduced benefits to allow affordability. Businesses may also see problems in recruiting employees if they do not offer attractive health benefits packages because some workers are in a "job lock," where they remain due to their company's health insurance coverage.⁶ Another tactic employers have taken is to form community coalitions on health, so that providers,

hospitals, and the employers have a voice in the purchasing of health care.

What are the alternatives?

This country has a fragmented and inconsistent system of delivery and payment for health care. Policy makers have looked outside of this country for potential solutions, particularly at systems in those countries which have an "universal health insurance" system or "single payer" system, in order to formulate policies that would allow increased and affordable access to health care. Reinhardt¹² states that countries have to decide how the provision of health care will be viewed, either as a "private consumption good" where each individual is responsible for paying, or a "social good" which is paid by all and utilized by all who need care, regardless of the ability to pay. The social good theory has been adopted by most European countries and Canada. The United States waivers back and forth. When we look at the public appropriations to the national health care expenditure per country for the Organization for Economic Co-Operation and Development (OECD) countries, the United States is by far the lowest. The U.S. public government share in total spending on health for 1992 was 45.7% compared to the combined OECD European countries' average of 78.5% (ranges were from 65.2% for Austria to 94.8% for Norway).¹³

Very few health care systems fit the classical definition of "socialized medicine," where the government owns, operates, and finances health care delivery. According to Reinhardt, the United Kingdom and Sweden qualify, as does the U.S. Veteran's Administration and armed forces. Most European countries have a mixture of private and public insurance, with purchase of private insurance mainly by the wealthy. About "90% of the population typically share one common level of quality and amenities in health care."¹²

England's system is predominantly a single source of financing for health care through national taxation, with care universally available and free at the point of service. General practitioners are the gatekeepers and there is rationing of services with waiting lists, especially for voluntary surgery.^{14,15} They utilize a needs-based formula for regional funding, taking into consideration differences in mortality, the age structure, and socioeconomic factors. In the early 1980s, specialty consultants steered the system and were not always considerate of efficiencies. During the late 1980s and early 1990s, a shift emphasized disease prevention and health promotion, recognizing a need to reallocate and better manage resources. This reform effort includes separating the payment from the provision of care, allowing hospitals to act autonomously, and positioning general practitioners at risk for services to the patient (similar to our gatekeeper system in HMOs).¹⁵

Rationing of services has been a frequent criticism of the British system, especially in relation to high-tech or life-extending services such as renal dialysis, coronary artery bypass graft surgery, and various cancer treatments. A part of the reform movement in 1989 included setting maximum waiting periods for procedures and making decisions regarding coverage of procedures/services based upon enhancing quality of life rather than merely extending life. In addition, the services should promote health and decrease disability.¹⁵

The German health care system is a social insurance model of government-mandated financing by employers and employees, and administration by not-for-profit agencies called sickness funds. The funds set and collect revenues and turn them over to regional associations of ambulatory care physicians, which in turn, reimburse the physicians for care rendered. Rates for hospitals and fee schedules for physicians are negotiated between the funds and the regional associations. The benefits are comprehensive to all citizens.¹⁶

The government finances most of the care for civil servants, with employees supplementing coverage through private insurance. Workers contribute a percentage of their gross salary (now 8% to 16%), which is matched by their employer. Workers making more than a set amount (~\$37,000) can opt out of the fund and purchase private insurance instead. Most elect to stay in the fund, but some of the well-to-do supplement the fund coverage with private insurance. Dependents are also covered.^{16,17}

Private insurance reimburses the physicians and hospitals much more handsomely than the funds, but "the majority (greater than 90%) of ambulatory care physicians are barred from treating patients in the hospital." Most of these physicians receive their income from treating the 88% of the population that have membership in the sickness funds. The cost containment efforts in this country include control of purchases of high technology equipment, increased scrutiny of physicians' practices, cost sharing, hospital budgets, decreased utilization of brand name drugs, and regulating physician fees.^{16,18} Physicians enjoy clinical autonomy in decision making, but give up their economic control to the professional associations that conduct the negotiations for them. Another interesting facet of the German system is a relative lack of research in health economics, health services, and epidemiology. Inglehart reports there are no disease-specific registries because of a reluctance of the citizens to have their names on lists, a result of the Nazi experience.¹⁸

Other European countries, such as Belgium, France, Ireland, the Netherlands, and Spain, basically have universal coverage for health care, compulsory insurance, pooled risks, and supplemental voluntary insurance. Their objectives have been to provide: access to basic levels of health care; payments related to the individuals' ability to pay rather than individual risk; health care expenditures that

consume an appropriate fraction of the gross national product (GNP); outcomes and satisfaction as high as possible; freedom of choice of providers and treatments; and autonomy for providers.^{13,19} The common problems these countries have faced are concerns with some groups not contributing to the pool of funds and increased health care expenditures without appropriate cost containment policies. Attempts to control these costs have included controlling the supply of services through planning, setting global budgets to help keep overutilization of services in check, and negotiating provider fees.¹²

Canada passed the Canada Health Act in 1984, which mandated coverage for all Canadian citizens without a copay. Income was no longer a barrier to receiving care and "the model was aimed at allocating resources made available for health care services in accordance with medical necessity (or the principle of 'reasonable access' to services)."^{20,21} This definition is clarified to stress the "need" of services and not the coverage of every possible service. There is rationing to some extent, as there is with all countries.¹² There are collective taxes which are distributed to the provinces if they comply with set requirements: universal coverage of the population; portability of coverage across provincial boundaries; comprehensive coverage of services; reasonable access to services without direct payment; and public administration of insurance plan.²⁰

Problems encountered with the Canadian system are not unlike those experienced in the United States. They are struggling with federal deficits and have gradually reduced the amounts of funding to the provinces, leaving the provinces focused on cost controls.²⁰ Although primary care services are readily available, there is rationing of specialty care by the physicians who make referrals.²² Some reports claim limited access to services, such as the United States having 3.26 open-heart surgery units per million people while Canada has 1.23 (cardiovascular disease is their number one health problem). This report also states that waits for urgent open-heart surgery can be eight weeks in Canada and that a patient is 10 times more likely to die waiting for surgery as during the surgery.²³

There have also been attempts to freeze the number of new physicians allowed to start practices due to an oversupply of practitioners and proposals to cut federal Medicare fees to new entry physicians in overserved areas. Other types of practitioners are providing primary care at lower costs, such as nurse practitioners and midwives. As a counter-move, physicians have now positioned themselves on important policy-making committees that have been responsible for setting practice guidelines to increase cost effectiveness of medical services. Physicians enjoy guaranteed employment and their incomes average about three to five times those of most working Canadians.²⁴ They also do not have to endure

Table 1. Comparisons of Life Expectancy and Health Care Expenditures

	At Birth**			At Age 65**			%GNP*
	total	men	women	total	men	women	
▼ France	77.1	72.9	81.3	18.3	15.7	20.4	9.4
▼ Netherlands	77.1	73.7	80.5	17.0	14.4	19.3	8.6
▼ Spain	76.4	73.1	79.7	16.9	15.0	18.4	7.0
▼ Germany	75.9	72.3	79.1	16.5	14.1	18.1	8.7
▼ United Kingdom	75.4	72.5	78.2	16.0	13.9	17.8	7.1
▼ United States	75.0	71.5	78.4	16.9	14.8	18.7	14.0
▼ Belgium	74.8	71.4	78.2	15.9	13.6	17.8	8.2
▼ Ireland	74.4	71.6	77.3	14.9	13.1	16.6	7.1

* % GNP of each country spent on health care¹⁰ (1992 data)

** Life expectancy data²⁸ (1990 data)

government intrusion and have little monitoring — they are the decision makers.

The comparison

When a single source payer system is promoted for the United States, policy makers attempt to compare our present system to those already in place that have developed some measures of outcome, both clinically and financially. Some of our recent health care reform efforts have called for global national budgets, as do other countries with universal coverage. Our country has been reluctant to accept such a concept because it appears to be against our free market system and there is always the question: "How much is enough to spend on health care?" Inglehart states the size of the disparity between a country's ability to hold the increase in spending for personal health services to a level approximating the growth of GNP indicates whether the system is operating in a socially acceptable fashion or if there should be efforts to slow down the growth of expenditures. This disparity has tipped toward costs outstripping the economic growth for all major western countries, with Germany being close to balancing the two in the 1980s and the United States having the greatest disparity. The United States has had a rate of growth of health care expenditures averaging 33% higher per year than the rate of growth of the national economy throughout the 1980s, compared with the 3% disparity in Germany.¹⁶ Only recently has there been a slowdown to the rise of health care costs — 7% in 1993 and 6.4% in 1994, which has been attributed to managed care and point of service plans.²⁵ Enrollees may be shouldering more of the burden for out-of-network care.

When deciding on an appropriate level of spending, one needs to look for excesses in supply or demand for health

services, and assess whether improved outcomes could be achieved if more resources are committed to "mainstream health services [and they] are not draining resources from other, more effective programmes."¹³ There is the need to conduct cost-benefit or cost-effectiveness analysis. The public needs to decide what they are willing to pay for health.¹⁴

One of the problems of determining the appropriate level of spending is that there are few scientifically proven benefits for most procedures and treatments, and there

exists a wide variation in practices. In addition, at this point, we spend the last dollar on what is perceived by us to be beneficial care to improve health or save lives, even if the benefit is marginal or unproved. We have not been sensitive to the true costs of health care because of the insulation provided by insurance.²⁶

Claims have been made that the problems of public financing and deficits will not be remedied by pumping in extra dollars without addressing the issue of efficiency in the delivery of care. Duplication of administrative efforts and costs throughout the health care system are usually attributed to multiple insurance companies with individual and small group policies, each with their own requirements and paperwork. McDermott claims that under single source payer systems, there are less administrative costs (<2%) compared to the United States (5% to 40%).²⁷ One report estimated that if the United States were operating under a Canadian health care model in 1991, there would have been administrative savings of \$46.8 billion. These savings may not have been realized as such because, with the single payer system, access to health care would have been provided to those without insurance causing increased costs in the provision of care.²¹

Inferences that health outcomes will be improved if we spend more on health care are yet to be proven. While the United States spends more for health care as a percent of GNP (14% in 1992 vs. 7% to 9.4%), the vital statistics for the population do not look the most favorable compared to the countries spending less. In a review of life expectancy at birth (Table 1), the United States ranks sixth among these countries, but if U.S. citizens live to age 65 years, then their projected life expectancy ranks third (a tie with Spain).²⁸ The disparity between these projections indicates that causes of death in the relatively younger populations have an in-

creased effect in the United States relative to the other countries that were reviewed.

Comparing the age-adjusted mortality rates per 100 000 (all causes) in these same countries, the United States ranks fourth highest (5th for men when stratified by gender and 4th for women). The mortality rates for specific diseases reveal the following ranks for the United States relative to the other seven countries:

■ respiratory cancer	1st
■ homicide	1st
■ motor vehicle accident	2nd
■ injuries/poisonings	3rd
■ circulatory diseases	4th
■ suicide	4th
■ malignancies	7th
■ lung disease	8th

Areas where we have relatively high mortality rates include respiratory cancers, homicide, motor vehicle accidents, and injuries/poisonings. This finding may indicate that we need to target specific areas of health to improve outcomes, rather than dumping money in an overall system. Care should be taken when trying to make inferences about the association of health care spending and outcomes, as prior data indicate a weak association due to other components that influence health, including social, environmental, and cultural factors.²⁸

How would reform impact our system?

We have attempted to control our spiraling health care costs with managed care programs, such as HMOs, and promoting utilization review programs. These programs have introduced the current problems of underserving patients, limiting patients' choices of providers, excess capacity of services and hospital beds, and much increased competitiveness. Although there is explicit rationing in the United States based on price and ability to pay, our country has been reluctant to promote implicit rationing as other countries do, where the services rendered are based on medical need and are ranked by physicians.¹²

Another obstacle our country has faced in making health care reforms is the political component. Many of the providers and owners of health care facilities, firms, etc., are business people making money off of the current system. The associations representing these groups often are well organized and have money to lobby politicians to maintain the system as is.²²

We have seen several attempts to reform our health care system with varying degrees of success. On a national level, Clintons' effort was stalled by the all encompassing nature of the proposal (too many changes with a rather vague conceptual framework), resistance by those that

are proponents of the current system (some health care firms and providers), and a secretiveness and exclusionary process of making decisions (the cabinet-level task force that Hillary Clinton chaired met in private without much public input and essential providers were not at the deliberation table).^{14,23,29}

On a state level, we have seen the much publicized Oregon plan, where rationing of care for Medicaid patients was highly debated, and finally accepted after many revisions. After utilizing a net benefit value formula, they "rank ordered health services according to their benefit to the entire population being served." This diverse group of decision makers came to a consensus as to coverage and ranked preventive services high on the list. This process was a demonstration of cooperation, where the public was involved, and cost-benefit analysis of a tremendous number of services/treatments was conducted to select an affordable benefits package.³⁰

Maryland also has introduced reforms on a fragmented basis. Legislation was passed regarding cost containment and small market insurance reform. Small market reforms have included limits on pre-existing conditions, guaranteed renewal of insurance, adjusted community rating, and requirements that all licensed insurers in the state offer a standard benefits package that is affordable.

If the United States were to introduce a form of universal health insurance coverage, some of the changes we could expect to see include:

1) an increased access to care, especially preventive care that can be promoted within the workplace, but not the traditional linkage between employment and health care coverage. All citizens would be covered with some type of basic health insurance which would alleviate the problem of the 42 million uninsured.^{1,2,4,27,29} There would still be underinsured patients, as every service would not be provided, because services are selected based on a cost-benefit analysis. There would be equal entitlements² and others would supplement their coverage with private insurance. This could set up a two-tiered system of health coverage, which most countries with universal coverage experience. There would be a shift of increased access to private providers by those not now covered. The public health providers may move more toward population-based care with abolition of the clinical public health infrastructure, which could have the effect of actually decreasing access to those who are not sufficiently motivated to seek care in private providers' offices.³¹

Barriers that exist today to receiving care would not be in play, such as pre-existing condition clauses, cost-shifting,³² exclusions, limitations, experience rating, and administrative overhead.²² If reform is conducted on a

national level, a much larger "risk pool" is created which would introduce cost-effective care.¹⁷

- 2) rationing of services so that we can afford basic coverage for all citizens.¹⁴ A benefit package would be assembled that includes services that have been selected through cost-effectiveness analysis.^{28,33}
- 3) uniform data collection systems put into place and run from a central location, so that more accurate information can be collected and analyzed to make policy decisions regarding provisions and coverage of care.^{27,33,34}
- 4) choice of provider along with continuity of care. This could be enhanced with increased preventive care rather than episodic visits for injuries/illnesses.²⁷
- 5) freedom in clinical decision making for physicians without unnecessary governmental or insurance company interference in the day-to-day practice.²⁷
- 6) savings by ridding unnecessary administrative overhead, such as processing claims, utilization review, marketing, determining eligibility, collections, data processing, and public relations.²¹

All of the above factors and the potential positive outcomes support the movement to introduce a single source payer system in the United States. There is strong opposition because of myths that have circulated, such as long waiting times to receive certain services (i.e., elective surgery). We would have to conduct a comprehensive and persistent educational program as to the merits and benefits the citizens will receive. The deliberations and discussions must involve all aspects of the public in an open format and emphasize selective coverage of benefits and not all encompassing services. With this approach, it is possible that we could develop a delivery system that would meet our needs for basic care and improve the health status of Americans.

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Tamoxifen induced endometrial abnormalities: Evaluation by saline infusion sonohysterography

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ABSTRACT: Patients receiving tamoxifen therapy for breast cancer are at an increased risk for endometrial hyperplasia, polyps, and adenocarcinoma. Therefore, any bleeding in the post-menopausal patient on tamoxifen warrants further investigation. Sonohysterography is a new technique for evaluating the uterine cavity and clarifying the location of endometrial and peri-endometrial processes. Differentiating subendometrial cyst formation from endometrial proliferation can save patients from further, invasive investigative diagnostic procedures.

A 60-year-old female presented for evaluation of post-menopausal vaginal bleeding. She had been diagnosed with Stage I infiltrating ductal breast carcinoma one and a half years prior, and had undergone lumpectomy with local radiation therapy. Additionally, she was receiving tamoxifen adjuvant therapy at the time of presentation.

The patient was evaluated via transabdominal and endovaginal pelvic ultrasound followed by sonohysterography. On transabdominal and endovaginal pelvic ultrasound, the uterus measured 6.6 x 6.6 x 8 cm. The endometrial stripe was grossly thickened (14mm) and heterogenous with cystic areas within and adjacent to it. During sonohysterography, the anterior endometrium was shown to be thickened to 5 mm, and posteriorly to 6 mm. Multiple cystic areas were also noted at the endometrial-myometrial junction.

The diffusely thickened and heterogenous endometrial stripe was consistent with cystic endometrial hyperplasia. In addition to endometrial changes, there was evidence of myometrial cyst formation at the endometrial-myometrial junction.



Figure 1. Sagittal image from endovaginal sonogram. Between arrows is thickened endometrium which appears echogenic. In the subendometrial area (curved arrows) are myometrial cysts.

Discussion

Tamoxifen is a recently introduced addition to the oncologists' armamentarium. As an adjuvant therapy for women with breast cancer, it has proven its worth in reducing five-year mortality in these patients.¹ Tamoxifen is a non-steroidal anti-estrogen agent which binds the estrogen receptor of tumor cells, inducing binding of the newly formed complex with DNA, therefore inhibiting receptor replenishment. This results in early, weak estrogen properties, followed by estrogen antagonism. However, studies have suggested that although tamoxifen acts in the breast as an estrogen antagonist, it occasionally functions as an estrogen agonist in the uterus, resulting in an increased incidence of endometrial hyperplasia, polyps, and adenocarcinoma,^{2,3} as well as adenomyosis⁴ and adenomatous hyperplasia.⁵ All of these entities may lead to vaginal bleeding in the post menopausal patient on tamoxifen.

Vaginal bleeding in the post-menopausal patient may have many causes, and an important distinction must be made between scheduled and unscheduled bleeding. The term 'scheduled bleeding' refers to cyclical bleeding in patients who are receiving hormonal replacement therapy, usually coincident with withdrawal from progestins. 'Unscheduled bleeding' refers to any other vaginal bleeding in the post-menopausal state which cannot be explained by phase of hormonal replacement therapy and progestin withdrawal. Unscheduled post-menopausal bleeding is an abnormal state associated with a variety of conditions, including senile endometritis, proliferative endometrium, hyperplasia, polyps, or neoplasia. In senile endometritis, the endometrium is atrophic with superficial bleeding ulcers, and is by far the most common cause of post-menopausal bleeding. Endometrial carcinoma accounts for only 7% to 30% of cases of post-menopausal bleeding.⁶ Further investigation of post-menopausal bleeding is warranted to rule out other causes, such as myomas, benign polyps, or endometrial hyperplasia. This is particularly important when evaluating post-menopausal bleeding in patients on tamoxifen, as they are at an increased risk for endometrial hyperplasia, polyps, and adenocarcinoma.

Ultrasound examination of the uterus can often detect endometrial abnormalities, providing the clinician with useful surveillance information for determining the appropriate course of action.

The atrophic endometrium of the post-menopausal patient has the sonographic appearance of a sonolucent, subendometrial zone, divided by a pencil sharp echogenic line of endometrium.⁷ Malinova studied 118 women and found a mean thickness of 3 mm in patients with histopathologically proven atrophic endometrium. They found no cases of endometrial carcinoma if the endometrial stripe was <6 mm.⁸ An endometrial stripe of >6 mm is suggestive of proliferative endometrium and warrants further evaluation. The Nordic Multicenter trial evaluated transvaginal ultrasound endometrial measurements in 1168 post-menopausal women, prior to curettage. They describe a 95% confidence of excluding endometrial abnormality (sensitivity 96%, specificity 68%) when endometrial thickness is less than or equal to 4mm by transvaginal ultrasound.⁹ It is therefore suggested that ultrasound be performed as a screening exam in patients at risk for endometrial carcinoma.¹⁰

However, it may be difficult for ultrasound to differentiate changes in the endometrium from subendometrial processes. Sonohysterography is a new technique for evaluating the uterine cavity which enhances endovaginal ultrasound examination by creating an endometrial fluid collection to better define the contour and thickness of the endometrial cavity.¹¹ A speculum is placed in the vagina and the cervix is prepped with Betadine solution. A preflushed, balloon hysterosalpingography catheter (5 Fr.) is inserted through the cervical os to the fundus, and the speculum is removed. The vaginal transducer is positioned in the vagina and, under ultrasound guidance, sterile saline is gently infused into the endometrial canal. Ultrasound scanning in both longitudinal and coronal planes is performed, with documentation on videotape or still image.¹¹

Sonohysterography has been shown to be a reliable method of clarifying location of periendometrial processes.¹¹ Achiron has shown that sonohysterography offers an accurate method of



Figure 2. Transverse (coronal) endovaginal sonogram shows thickened endometrium (between arrows). Cystic areas (curved arrow) appear within, and adjacent to, the endometrial stripe.



Figure 3. Sagittal image during early filling phase of sonohysterography. Balloon catheter is identified in the lower uterine segment (straight arrow). With suboptimal distention the endometrial canal is not well visualized. Subendometrial cysts are identified (curved arrow).

distinguishing endometrial polyps from an irregular endometrial-myometrial junction in 20 patients on tamoxifen.¹² Goldstein describes five patients receiving tamoxifen therapy, who were found by endovaginal ultrasound to have abnormal appearing endometrial canals.¹³ Endometrial biopsies in these patients revealed inactive endometrium. In all five patients, subsequent sonohysterography showed the irregularities to be beneath the endometrium, in the proximal myometrium. These changes were felt to represent adenomyomatous-like changes. Hysterectomy in one case revealed reactivation of foci of adenomyosis in the form of myometrial microcysts. Prolonged estrogen-like stimulation of the uterus by tamoxifen has been associated with a three to four times increased rate of adenomyosis in breast cancer patients, as compared with accepted rates in pre- and post-menopausal women not treated with tamoxifen.¹⁴



Figure 4. Sagittal image obtained during sonohysterogram, with catheter noted in lower uterine segment. With better distention of the uterine cavity, detail of the thickened endometrium is easily distinguished. (Cursors demarcate the width of the anterior and posterior segment of the endometrium.) No polyps or myomas are identified. Uniformly thickened endometrium is most consistent with endometrial hyperplasia.

In conclusion, patients undergoing tamoxifen therapy are at increased risk for endometrial hyperplasia, polyps, and adenocarcinoma. However, many patients will develop subendometrial cysts without endometrial proliferation. Management ranges from observation to surgical intervention; therefore, non-invasive techniques to screen patients on tamoxifen are essential. Sonohysterography plays an integral role in differentiating these processes, saving patients from further invasive investigative procedures.

Follow-up

Several months after her sonohysterogram, this patient underwent endometrial biopsy. Histologic examination demonstrated no evidence of malignancy.

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Chorea gravidarum: A case report and review

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ABSTRACT: A 23-year-old pregnant Pakistani female presented with hemichorea and hemiballismus at six weeks gestational age. Similar symptoms had occurred during a previous pregnancy resulting in a spontaneous abortion. Chorea gravidarum, a disorder characterized by choreiform and athetoid movement presenting during pregnancy, is rare. In the past, rheumatic disease was generally the etiology, but today, collagen vascular disease should also be considered. Treatments include neuroleptics for symptomatic relief and therapies targeted toward the underlying pathology.

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Chorea gravidarum is a rare neurologic occurrence that should be considered a symptom of underlying central nervous system pathology rather than a diagnosis. Chorea is a rapid, primarily distal, nonstereotyped movement. Slower, writhing, athetotic movements may intervene. These symptoms may present at any time during pregnancy, and even with maximal treatment may progress to a hyperthermic, hypermetabolic state that can be fatal to both the fetus and the mother. Recognition of the underlying pathology allows the physician to proceed with appropriate therapy and avoid unnecessary, potentially dangerous diagnostic evaluations and treatments.

This article describes a patient with chorea gravidarum who responded well to symptomatic treatment and proceeded to a normal delivery with a healthy, full-term infant. The sad and serendipitous history of chorea gravidarum is discussed. The pathophysiology and treatment options are also reviewed.

Case Report

A 23-year-old, right-handed, pregnant Pakistani female presented to the emergency department at six weeks gestational age with the acute onset of uncontrollable, left upper extremity movements that had begun in the morning. Throughout the day, these movements had progressed from twitching of her fingers to constant, choreoathetoid activity punctuated by violent hemiballisms that had already resulted in several bruises and a laceration to her left wrist. Four years before, the patient had experienced similar problems during the first trimester of another pregnancy. At that time, her symptoms worsened over several days until she miscarried, at which point they abruptly ceased. In the interim, she had an uneventful pregnancy producing a full-term, normal male. The sex of the aborted fetus was not known. She denied headaches, weakness, numbness, fever, trauma, or recent infections. Later conversations with her mother revealed that, at five years of age, the patient suffered from acute rheumatic fever followed by severe Sydenham's chorea, limited to the left side, which took several months to completely resolve.

Her initial exam was significant for a slightly thin female lying in bed with constant choreoathetosis in both the left upper and lower extremities. The arm also had intermittent, violent abduction that was limited only by restraints. Her face contorted occasionally, which her husband noted had also begun that morning. She was afebrile, with a blood pressure of 105/70 mm Hg and a heart rate of 98 beats per minute. Her general examination was remarkable only for a gravid uterus and the bruises and abrasions noted above. There was no evidence of valvular disease, alopecia, rashes, or nuchal rigidity. Neurologically, she was awake, alert, and completely oriented. Although she was appropriately distressed, she was not emotionally labile and had no evidence of hallucinations or paranoia. Speech and language were intact except for slight hesitance associated with facial grimacing. Cranial nerves were intact. She had 5/5 strength but was unable to suppress the movements enough to cooperate fully. Deep tendon reflexes were normal and plantar reflexes were in flexion. On the right side, she had normal fine motor function, normal rapid alternating movements, and no dysmetria. Sensation was intact to all modalities. Tone was normal. Her gait was slightly unsteady due to left foot athetosis.

Her only medications were a multivitamin and an iron supplement, which she had been taking for one week. Both of her parents and several siblings were alive and well with no neurologic or psychiatric problems. Laboratory values were

notable for normal electrolytes and liver functions. See **Table 1** for other laboratory data. A cardiac echogram was normal.

She received parenteral chlorpromazine 50mg intravenously and within an hour the hemiballisms had stopped. She refused admission and was discharged with a prescription for chlorpromazine 25mg tablets. As an outpatient, the dose was gradually increased to 50mg every eight hours. Ten days after her initial presentation, only subtle choreoathetosis on the left upper extremity with arm abduction was evident. However, she noted that if she missed a dose of the chlorpromazine, the movements resumed with a fury. Over the next 10 weeks, she continued the chlorpromazine and then gradually tapered off as her symptoms subsided. By the beginning of the third trimester, she was asymptomatic off medications with a completely normal neurologic examination. At 38 weeks gestational age, she vaginally delivered a healthy male.

History

In 1932, Wilson and Preece published an extensive review of 951 cases of chorea gravidarum—the largest to date.¹ They estimated the occurrence to be approximately 1:3000 pregnancies and noted a maternal mortality rate approaching 30%. If the pregnancy was allowed to proceed, there was a 50% fetal mortality rate. Among their patients, over 86% had a history of rheumatic heart disease, leading them to hypothesize that the movements were an autoimmune phenomenon.

This was a major breakthrough as previous theories attributed chorea gravidarum to "psychic conflict" in reluctant young mothers. Hysteria and illegitimacy were also blamed. A "cervical reflex phenomenon" was described to account for the movements. Elaborate treatment schemes had evolved,¹⁻³ many of which undoubtedly increased both the fetal and maternal morbidity and mortality. Sedation with paraldehyde, barbiturates, chloral hydrate, or morphine were used. Medical student relays were sometimes formed for the

Table 1. Laboratory data

Laboratory	Result
sedimentation rate	8
peripheral smear	normal-no acanthocytes
rheumatoid factor	negative
anticardiolipin antibody	negative
antiphospholipid antibody	negative
anti-streptolysin antibody	positive at 166
thyroid stimulating hormone	1.0
anti-nuclear antibody	1:160 speckled

Table 2. Other reported causes for chorea gravidarum

- ▼ vascular malformations
- ▼ cerebrovascular accident
- ▼ thyrotoxicosis
- ▼ Wilson's Disease
- ▼ Huntington's Disease
- ▼ neuroacanthocytosis

continuous administration of chloroform. Such efforts frequently resulted in overdose and respiratory arrest. Bizarre therapies with cervical iodine preparations, tonsillectomy, restrictive diets, and parenteral horse serum were tried, as were arsenic compounds and potassium bromide. If these measures failed, "therapeutic" abortions (with a 34% mortality rate) were often recommended. In 1956, Winkelbauer and Kimsley⁴ serendipitously discovered the efficacy of chlorpromazine when they gave a parenteral dose to a chorea gravidarum patient for nausea and vomiting. Within 30 minutes, the patient's movements resolved and continued to respond to subsequent doses.

Natural History

Chorea gravidarum may present at any time during an otherwise uneventful pregnancy and can be preceded by psychiatric symptoms ranging from emotional lability to psychosis. Some reports suggest that the incidence is higher in primiparous women, but in the past, there was obviously a selection bias since patients frequently did not survive to a second pregnancy. In severe cases, the movements may progress to hemiballisms, self-injury, rhabdomyolysis, hyperthermia, and death. Movements subside in sleep. Even mild cases may prevent the patient from performing activities of daily living. Today, the overall mortality is estimated at <1%.⁵ The chorea almost invariably stops within hours after delivery. Etiology determines time of onset, prognosis, risk of recurrence with subsequent pregnancies, and potential treatments.

Etiology

Rheumatic Disease. Most early reports of chorea gravidarum were likely related to rheumatologic disease, but since the advent of antibiotics, the incidence of chorea gravidarum has declined. These patients frequently have a history of recurrent tonsillitis or childhood Sydenham's chorea.^{1,6} Symptoms usually present in the first trimester and abate late second or early third trimester. Imaging studies are

usually normal,⁷ but presumably, previous rheumatic disease has caused basal ganglia or caudate nucleus defects which become symptomatic in the presence of increased estrogens. Dopamine hypersensitivity occurs due to post-synaptic modifications in high-estrogen states.⁸ The relationship between increased estrogen and chorea gravidarum became evident in 1975 when oral contraceptives, with much higher hormone content than today, became widely available and resulted in an epidemic of chorea in young women.⁹

Antistreptolysin antibodies are elevated and may continue to rise during the pregnancy. Cardiac valvular disease is often present, and therefore, prophylactic antibiotics may be needed during delivery. Patients generally do well, but there is a 25% recurrence rate.¹

Autoimmune Disease. Systemic lupus erythematosus (SLE), anticardiolipin antibodies, and antiphospholipid antibodies are the more prominent causes of chorea gravidarum in industrialized countries today.¹⁰⁻¹⁴ Presenting in the second or third trimester with agitation and confusion, these patients are more likely to proceed to rhabdomyolysis, seizure, hyperthermia, hemiplegia, coma, and death.^{1,2,5,11} They frequently have a history of fetal loss. Imaging studies can show focal changes in the caudate nucleus and basal ganglia, presumably due to infarction or vasculitis.^{10,11} Cerebrospinal fluid reveals a pleocytosis and an elevated protein. Occasionally, chorea may persist after delivery. Recurrence with subsequent pregnancies has been reported with fatal results.^{1,15}

Less common etiologies may need to be investigated if neither of the above are obviously the cause. These are listed in **Table 2**.^{7,9,3,16-18}

Treatment

For symptomatic relief in patients with chorea gravidarum due to rheumatologic disease, phenothiazines are the mainstay of treatment during the first trimester. These drugs are class C during pregnancy,¹⁹ but significant experience in their use for hyperemesis gravidarum suggests little teratogenic effect. Haloperidol is less sedating and may be used in the second and third trimesters, but reports of limb deformities prohibit first trimester use.²⁰⁻²²

Patients with SLE as the cause of chorea gravidarum should receive immunosuppression via steroids.^{10,12,16} Aspirin therapy in cases of anticardiolipin antibody and lupus anticoagulant is also indicated.^{13,14} Neuroleptics may also provide symptomatic relief, but the underlying pathology must

be addressed to prevent irreversible damage. Obviously, therapeutic abortions are a last resort, but should be considered in especially severe cases resistant to other treatment.

Conclusion

Chorea gravidarum is a rare disorder that should be considered in all women of reproductive years presenting with acute chorea and/or athetosis. Though potentially a morbid entity, treatments which address the underlying pathology are available if the etiology is known. Historically, medical treatment probably worsened outcome due to grim therapies with high complication rates. Avoiding unnecessary, aggressive diagnostic evaluations is important to prevent history from repeating itself.

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What to call for

Urinary tract infections (UTIs) are one of the most common disorders prompting a physician visit, second only to upper respiratory tract infections. Most of these infections occur in otherwise healthy young women and girls with normal urinary tracts and normal urinary functioning, and are usually considered uncomplicated infections. A survey by the American Medical Association (AMA) revealed that women were not knowledgeable of the cause of the infections, nor of ways to prevent such infections. The AMA has published a patient education booklet, *Urinary Tract Infections: A Patient's Guide to Treatment*. Physicians can obtain copies of this pamphlet for distribution to patients, by calling 312-464-2588.

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MARYLAND MEDICAL HISTORY

Celeste Lauve Woodward, M.D.

In the last half century, the presence of women in medical schools has increased from just a few per class to roughly 50%. An outstanding pioneer who trained in the days when medicine was essentially a “man’s profession” is Celeste Lauve Woodward, M.D., the wife of Theodore Woodward, M.D., himself a leading international medical figure.

Celeste Lauve was born in Upper Montclair, New Jersey, in 1912, the fourth of five children. She had three older brothers – Louis, Jr., Henry, and Omer – and a younger sister – Anita. Celeste’s father, Louis, was born in Galveston, Texas. Because his parents died when he was only nine, he and his older brother were sent north to one of the few boarding schools for boys then in existence – Georgetown in Washington, D.C. There Louis met his future wife and Celeste’s mother, Constance McCullough.

Constance was restless and wanted to travel, see Europe, and learn more of its languages and cultures. The Lauve family left the United States in 1925, when Celeste was 12 years old. She and her siblings were enrolled in boarding schools in France. Her parents, then living in Paris, had planned to remain in Europe for only one year but remained for nine, primarily residing in France, Switzerland, and Austria.

Dr. Woodward’s first interest in medicine probably dated from her observation of the effects of many mastoid operations performed on her five-year-old brother and her mother’s influenza (complicated by pneumonia and empyema in 1917), resulting in a stay in Charlottesville, Virginia. In Charlottesville, she also witnessed the preparation of a medical unit at the University of Virginia Hospital prior to embarkation during World War I.

She was interested in obtaining her bachelor’s degree in France in the mid-1920s, a time when the only option for women was tutoring at boarding schools. The “premiere partie” (junior year) required written and oral examinations in all subjects. She completed this in 1931 in Paris at the Sorbonne. For her “deuxieme partie” (senior year), she was tutored in Nice, where she received her bachelor’s degree at the Universite d’ Aix-Marseilles “Mention bien” (cum laude) and was awarded a scholarship to the Universite de Lyons. However, because they did not admit women, her application to their medical school was rejected.

In 1934, the family returned to the United States, locating in Baltimore, where her father had accepted a sales position. Celeste applied for admission to The Johns Hopkins School of Medicine, but they had already filled their openings for that year. She was advised by the dean, Dr. Alan Chesney, to spend the year obtaining a master’s degree, and to apply the following year. Because she felt that she had “already wasted too much time,” she instead sought admission to the University of Maryland School of Medicine.



Dr. Celeste Woodward in her early days of practice.

She was accepted to the 1934 freshman class. As fate would have it, her future husband, Theodore, was also enrolled in that class. However, Dr. Woodward notes that she didn't start dating him until their junior year. They graduated in June 1938 and were married about three weeks later. She began her internship the next week at the Baltimore City Hospital (now Johns Hopkins Bayview Hospital).

Dr. Woodward's husband was called to active duty in early 1941 and sent to Walter

Reed Hospital in Washington for training in tropical diseases. Upon completion of his instruction, he was ordered to Bermuda and then to Jamaica, where she planned to accompany him. Unfortunately, after Pearl Harbor, the Army issued an edict that families could no longer accompany their spouses.

While busily engaged in rearing her children, Dr. Woodward worked for a short time at St. Agnes Hospital which was acutely short of medical personnel. She then was employed by the Baltimore City Health Department as a quarantine officer covering one quarter of the city. In those days, communicable diseases were of frequent incidence. An important measure in the prevention of spread was strict isolation. Appropriately for this era, two of her sons came home with pertussis, which she promptly caught and passed on to Craig, her third son. She worked as a volunteer physician during World War II in the medical clinic, first in the original (1823) Baltimore Infirmary at Lombard and Greene streets — then in the clinic in the "new" University Hospital.

In 1955, her husband traveled to the Orient to lecture in Japan and other Asian countries. William, Lewis, and Celeste accompanied him. While in the Philippines, their second son, Lewis, not yet 15 years old, suddenly expired from an unexplained cardiac arrest. When her husband returned to the Orient in 1961, this time to what was then West Pakistan, he was impressed with the acute need for primary care

On her first trip to Thailand, Dr. Woodward was informed that she would be required to have her medical school diploma with her in Bangkok to verify that she was really a physician. Dr. Woodward was reluctant to carry this certificate with her, as she feared it might be irretrievably lost. Luckily, she and her husband were scheduled to have dinner with the dean of the medical school that evening and she happened to mention her problem. The next day he provided her with an exact duplicate of her original sheepskin (quite a feat since photocopies were yet to be invented). Dr. Woodward believes she is "the only physician to possess two original medical diplomas."

physicians and dermatologists. He suggested that his wife seek additional training in the diagnosis and treatment of skin diseases. She gained this experience over a two-year period at the Dermatology Clinic of the Robinsons at her alma mater. Later, her husband took a sabbatical from his position as head of the department of medicine at the university and she accompanied him. She taught at the Fatima Jinnah Medical School, seeing patients in the Sir Ganga Ram and the King Edward clinics

in Lahore, West Pakistan, and at the same time making a survey of the incidence of skin disease for the National Institutes of Health in Bethesda, Maryland.

During her stay in West Pakistan, she was asked to see one of the most influential men in the country who had a skin condition which had been resistant to the therapies local physicians had used. His servants who called for her, not knowing that she understood some of their language, spoke among themselves in derogatory terms that "any woman" should be consulted in such a situation. She found that much of the patient's difficulty was due to over-treatment; the use of conservative therapy improved his condition. The servants' change in attitude became obvious as they began treating "this woman" with utmost respect.

While in Pakistan, Dr. Woodward saw much leprosy in all stages, as well as most of the tropical diseases rarely encountered in our country. She believes these also included some of the last cases of smallpox seen in the world.

Workers tending the sick indicated the need to separate the terminal cases of leprosy from the earlier ones so that the latter group would not be regularly viewing their likely ultimate plight. Since her very wealthy patient had built many charitable structures and endowed many worthy causes in the country, she attempted to use her influence on him to provide for this need. He promised her that he would give it serious consideration but, unfortunately, succumbed to a

heart attack before he was able to act.

In 1968, under the auspices of the American Medical Association (AMA), Dr. Woodward joined a group of 11 other physicians and eight nurses who traveled to South Vietnam to an area northeast of Saigon, known as Nha Trang. She was there for five months, during which time there was active fighting in the area. She could hear machine guns firing from the equivalent of about a block away. The Viet Cong had wiped out entire villages, and their hospitals were so crowded with civilian casualties that two or more people occupied every available bed. Dr. Woodward made a habit of having breakfast each Sunday morning with the Green Berets. On one occasion, the jeep in which she traveled was found riddled with shrapnel holes made while she was having her morning meal.

In 1970, she returned to Vietnam, this time assigned to an area in the Central Highlands known as Bao Loc. The area was surrounded by mountains, and with the absence of modern radar or other sophisticated equipment, it was too dangerous for airplanes to land. Therefore, she was carried in and out by helicopters. On one occasion, not long after she had flown on one, it crashed killing all aboard. During the four months in Bao Loc, Dr. Woodward treated as many cases of leprosy and plague as she did casualties of war and malnutrition.

Also, beginning in 1968, when she was in Baltimore, Dr. Woodward, as assistant professor of medicine, volunteered her time in the emergency room of the University Hospital. This continued for 13 years, but in the mid-1970s, the administration felt it could no longer accept her work gratis and they agreed upon a minimal token salary.

Dr. Woodward looks back at those days with some nostalgia. She recalled in that era the other general hospitals mostly sent the alcoholics, prisoners, and chronically and mentally ill to the University Hospital (then a state hospital). Also the residents who were assigned to see them looked upon this work as an uninteresting chore. Dr. Woodward noted the wealth of pathology these cases presented and stressed this opportunity to these young physicians.



Dr. Celeste Woodward holds a model crafted for her by a refugee whom she befriended while in Thailand.

She indicated, however, that the job was not without risk. On one occasion, when she was asked to see a prisoner from the prison, the individual requested that he be allowed to use the rest room. This required that the guards remove his manacles. As one of the guards was performing this task, the prisoner attacked him, grabbed his gun, and pointed it at the other guard and everyone else within range. Fortunately, before the weapon could be fired, the second guard was able to dislodge it from his hand. No one was injured.

Dr. Woodward's fourth and fifth trips to Asia as a treating physician were to Thailand – the first in 1979, the second in 1980. These were made under the auspices of the American Refugee Committee of Minneapolis. (The unpopularity of the Vietnam War prompted the AMA to discontinue earlier sponsorship.) On the first trip to Thailand, she was sent to the northeast portion in an area known as Ban Vinai, only eight kilometers from the Laotian border. There had been a primitive hospital previously operated by the French Doctors Without Borders. The medical team was comprised of four physicians and eight nurses. Dr. Woodward saw many cases of scabies as well as conditions she had treated elsewhere in Asia. In Ban Vinai, she was even called upon to extract teeth. During this tour in Thailand, she contracted dengue fever.

Between the monsoons and the defective plumbing, the area was like a shallow river with discharge from the outhouses evident everywhere. Despite what in our eyes might suggest a place to be avoided, as soon as the floods receded, the area exploded with refugees from Laos, with the population jumping from 5,000 to 45,000. Many of these were people of Chinese descent who had lived in the mountains of Laos and were being sought out by the communist Pathet Lao Army. They were known as Hmongs and were intelligent and industrious people. One of these was a 24-year-old cook who operated a tiny restaurant. The communists had placed a price on his head because he had been a spy. He attached himself to Dr. Woodward as he wished to learn English. Eventually, she was able to obtain permission

for his emigration to the United States and even made tentative arrangements for his employment in Baltimore. However, immigration authorities insisted he be sent to Kansas City where his uncle resided. He became a chef at a country club, but found he missed his homeland and returned to Asia. She still has in her possession several beautiful pieces representative of Asian structures which he crafted.

Dr. Woodward's last trip was to the southeastern area of Thailand bordering Cambodia. The group occupied a 90-plus acre camp site, again receiving and treating refugees. On this occasion, they consisted of both North and South Vietnam Army personnel who were absent without leave, as well as Cambodians fleeing from the Pol Pot and the Khmer Rouge. All were masquerading as peasants; if they were apprehended and believed to be otherwise, they would have been shot. Some of the early refugees presented with high fevers not immediately recognized as cases of measles. When Dr. Woodward discovered they faced a potential measles epidemic unless others were immunized, she contacted authorities in Bangkok. The vaccines did not arrive for three months; a measles epidemic was in full swing.

On one occasion, Dr. Woodward was taking a bus out of the capitol city and noted a young lady taking pictures of all the passengers. She thought the photographer wanted to sell them and she was anxious to purchase one. To her surprise, the lady refused her request and left the bus. Dr. Woodward later discovered that on numerous occasions bus passengers had been robbed. Highwaymen, in cahoots with a passenger, robbed the bus when it reached a predesignated and isolated area. The photographs were being taken to identify the thieves. From that point on, Dr. Woodward chose to fly.

She shortened her Thailand stay by a month in order to be with her daughter, Celeste Applefeld, who had recovered from a serious illness and was about to deliver her second child.

While her father-in-law, Dr. Lewis K. Woodward, Sr., had been a physician in the general practice of medicine, and

Dr. Celeste Woodward tells the story of one morning going into her living room and finding her husband saying to one of their younger grandchildren, "Sign this paper." She immediately jokingly advised the child not to sign her name to anything without the advice of a lawyer. At the same time, she looked over her husband's shoulder and read the note. It stated, "I will be a pediatrician." To her surprise, the child took the paper and signed it. However, when she again viewed it, she had signed her brother's name to it.

her brother-in-law, Dr. Lewis K. Woodward, Jr., had been a surgeon, the only medical individual on the Lauve side had been a Northern army surgeon sometime prior to the Civil War. However, all three of her living children – William, Craig, and Celeste – are physicians. The elder son has a vast background with a bachelor's degree cum laude from Princeton, a medical degree from Hopkins, and an internship at Vanderbilt. He

joined the U.S. Public Health Service as a public health officer specializing in diseases which produced diarrhea. Because of his background, he was sent to East Pakistan where he worked with cholera for two years, and then he worked in other areas of need worldwide. He continues to utilize his knowledge for the public good by volunteering at hospices and by other community efforts. Her second surviving son is an internist in Atlanta, also boarded in cardiology, on the staff of Emory University and part of a four-physician practice. He was one of the physicians for the recent Olympics. Her daughter Celeste is a pediatrician and a professor of pediatrics and medicine, associated with Mercy and University Medical Systems. Her son-in-law is also a cardiologist and chief of cardiology at Mercy. The Woodwards have nine grandchildren ranging in age from 8 to 30. While none are physicians yet, they feel several may be.

As one reviews the life of Dr. Celeste Woodward to date, one must conclude that despite personal and family illness and tragedy, she has contributed immeasurably to the well-being of innumerable human beings, not only in Baltimore but the world. The risks of exposure to exotic diseases or to injuries or death from wars and other forms of mayhem did not deter her. Since there has been so much accomplished medically by her husband, Dr. Theodore Woodward, by her in-laws, and by her children, she has been content to call little attention to herself and her accomplishments. May she, and they, be with us for many years and may they continue their good works. They are a beacon for all.

MARION FRIEDMAN, M.D. ■

MARYLAND MEDICAL HISTORY

Helen Brooke Taussig, M.D.: The original pediatric cardiologist

Helen Brooke Taussig, M.D., the original pediatric cardiologist, died instantly on May 20, 1986, as a result of an automobile accident, which occurred three days before her 88th birthday. She apparently did not see an approaching car as she turned from a parking lot to a main road.

While she will be most remembered for the Blalock-Taussig operation, which prolonged the lives and usefulness of thousands of children and opened the door for other forms of pediatric cardiac surgeries, her impact on our world was much greater. Dr. Taussig was born in Cambridge, Massachusetts. Her mother died when she was eight years old, but her father, brother, and sisters, and their families, maintained close ties throughout her life.¹ Personally and professionally she had many difficulties to overcome. Besides the death of her mother, she overcame dyslexia and a hearing impairment.²

Her undergraduate studies began with two years at Radcliffe College and were followed by another two years at the University of California, where she graduated Phi Beta Kappa in 1921. She attended Harvard Medical School as a special student, but her gender prevented her from matriculating as a degree candidate. After this, she spent time at Boston University School of Medicine, where she developed an interest in the anatomy of the heart. She transferred to the Johns Hopkins University School of Medicine and graduated in 1927.³ Although Dr. Taussig originally desired a fellowship in medicine at Hopkins, she was instead assigned to their heart station. This led to an internship in the Hopkins' pediatrics department. After completing two years of study,

she was offered a position as physician-in-charge of a newly created Harriet Lane (Pediatric) Cardiac Clinic.⁴

After she became hard of hearing, she sometimes characteristically made a virtue out of necessity by using her hearing aid to make her presence and opinions felt. Usually sitting in the front row at meetings, she would at strategic points adjust the instrument with much screeching and then turn it off—more out of disapproval at what was being said than with the instrument. Yet, in discussion and argument she was always polite and to the point with a good sense of humor.

—The Lancet, July 12, 1986

Early on, she came to believe that cyanosis in children was due to inadequate oxygenation of the blood in the lungs. About that time, she learned of the successful closure of a patent ductus arteriosus in Boston. Her hypothesis and the new developments in cardiac surgery, led her to believe that this approach could favorably alter the then dismal prognosis for cyanotic children. She presented this theory to Dr. Edward A. Parks, then chief of pediatrics, and Dr. Alfred Blalock, then chief of surgery.⁴ Operating techniques were developed in association with Vivien Thomas, an African-American technician in charge of animal surgery. The first Blalock-Taussig operation was attempted after it was performed on about 200 dogs.³

While the initial operation to correct the defect was a failure,

three subsequent attempts were successful. The third operation was performed on a small six-year-old boy "who was utterly miserable and no longer able to walk," had intensely blue skin, and deep purple lips. After the final sutures were tied and the clamps released, the anesthesiologist gleefully called out, "The boy's a lovely color now." Over the ensuing years, there has been an 80% success rate.⁴ Subsequent to this successful research effort, Vivien Thomas was awarded an honorary doctorate degree.

Drs. Blalock and Taussig reported the results of the first three successful operations in the *Journal of the American Medical Association* in May 1945. They commented that "there had previously been no satisfactory treatment for pulmonary stenosis." The journal captioned this a "Landmark Article."⁵

Once the success of the Blalock-Taussig operation was publicized, there was an immediate increase in the referral of patients with congenital heart defects to pediatric clinics all over the country. Pediatric cardiac clinics were established in academic centers throughout the United States.⁶ As word spread of the new hope for blue babies, patients began to deluge Dr. Taussig's cardiac clinic, arriving from all parts of the country and abroad.³ Dr. Richard J. Bing tells of receiving a call from Dr. Blalock to join him in setting up a laboratory for the physiologic and diagnostic study of congenital heart disease. His initial trip to Hopkins required him to take a ferry, which then was the usual method of crossing the Chesapeake Bay. Dr. Bing encoun-

At a service attended by more than 175 doctors, former patients, and admirers, one Mary Walker, 49, Taussig's 263rd blue baby patient, said doctors had told her parents that she would not live beyond the age of 13. "To this great lady I owe my life," said Walker, who underwent Taussig's life-saving operation.

Walker recalled the moment, some 40 years prior, when she awakened from surgery: "My grateful parents were on one side and Dr. Taussig was on the other, holding my pink hand."

—*The Evening Sun, June 27, 1986*

Taussig that children with congenital heart disease can now be treated adequately in most European countries, that a cardiac institute is flourishing in New Delhi, and that cardiac research was given a new impetus.⁹

One biographer³ felt that Dr. Taussig's principal contribution to medicine was her education of other physicians. In 1944, there were only seven pediatric cardiologists in the United States. By 1963, she had personally trained more than 60 physicians practicing this specialty in the United States, and a number from foreign countries. Many have become professors of pediatric cardiology, with their own training programs in institutions such as Yale and Baylor.

Over a quarter of a century prior to her death, a child with a virtually hopeless heart defect came to Baltimore for an operation supervised by Dr. Taussig. The day before the operation she invited the parents to her home and spent an entire afternoon counseling and consoling them, but never minimizing the danger. The child died in the operation, but Helen Taussig stayed in touch with the parents for the rest of her life. This is the kind of human being she was.

—*The Evening Sun, May 23, 1986*

tered at least six cyanotic subjects on this short boat ride, not all of whom were children.⁷

Staff and space had been adequate, but one secretary, one electrocardiogram (ECG) technician, one social worker, and two fellows, together with Dr. Taussig, were now suddenly overwhelmed by the onslaught of patients.⁸ Fellows and visitors came from the Americas, South Africa, India, and Europe. It is thanks to Dr.

She devoted ten years to writing her classic book, *Congenital Malformations of the Heart*. It immediately became the "bible" for all those with an interest in the new and challenging fields of pediatric cardiology and cardiac surgery.¹⁰

Dr. Taussig was one of the six members of the first certifying board of pediatric cardiology¹¹ and the first woman president of the American Heart Asso-

ciation (AHA).¹² While president of the AHA, she publicized her conviction that atherosclerosis begins in infancy and childhood.⁷ She was also the first woman to be admitted to the American College of Physicians. In 1973, they established a Helen B. Taussig Lectureship.¹³

Despite her innumerable contributions to pediatric cardiology, her humanitarian interests extended beyond the boundaries of her own specialty. In the early 1960s, when birth defects began to appear in the offspring of European mothers who had taken the drug Thalidomide, she went to Germany to study the problem. She then promptly published warnings to help prevent a similar epidemic in the United States.³ Dr. Taussig was also instrumental in obtaining the passage of legislation by Congress mandating the careful testing of pharmacological agents used during pregnancy.¹¹

Her frugal personal habits carried over to her national service, making her interested in the economics of health care delivery. An example of her disdain of waste was noted when she traveled to Paris in 1947 to be made a Chavalier of the Legion d'Hommeu. It was reported that she was "scandalized" when the U.S. Embassy placed a car and driver at her disposal for several days.³

In 1952, she reported on the long-term follow-up results of the first 1000 Blalock-Taussig operations. After her retirement, she analyzed these same cases, becoming a pioneer in the use of the computer for such purposes.³

Even though Dr. Taussig was at Hopkins from the time she received her medical degree until her retirement in 1963, some 36 years, she was not awarded full professorship until four years prior to her retirement. Nevertheless, Dr. Taussig remained actively involved in research over the next 23 years prior to her sudden and untimely death. During these last years, she lived in Kennett Square, Pennsylvania, and drove herself weekly to Wilmington's Museum of Natural History to study heart malformations in wild birds. She learned from her studies and those of others that birds, reptiles, and mammals have cardiac

Three months before her untimely death, she attend a recital in her honor by one Samuel Sanders, who 39 years earlier had undergone the operation developed by her and Dr. Alfred Blalock.

—The Evening Sun, May 21, 1986

malformations similar to those seen in children. She completed her last manuscript only a few days prior to her death.¹²

Dr. Taussig received innumerable honors, including 20 honorary doctorate degrees (18 DSc, one LLD,

and one Doctor of Humanity) and over 30 medals and awards. At least ten of these were bestowed by foreign nations.¹⁴

Even after she could no longer actively assist in alleviating problems in the world, she did the only thing she could — she donated her body to Hopkins.⁴

MARION FRIEDMAN, M.D.

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Book review editor:
Chris Papadopoulos, M.D.

Remembrances of Thirty Years in Baltimore. Lilian Welsh. Baltimore: Norman, Remington Co.; 1925. 167 pages. Available at The Johns Hopkins Welch Medical Library, the Med Chi library, and the Goucher library.

This book is a must read for anyone interested in the history of medicine in Baltimore and Maryland, or the history of women in medicine.

Ever had to choose between becoming a doctor or a lady? This was the dilemma expressed by the daughter of famous Hopkins obstetrician Dr. Howard A. Kelly when asked by Dr. Lillian Welsh if she would like to be a doctor like her father — the young Miss Kelly was adamantly on the side of becoming a lady. Dr. Welsh records this anecdote about the social and educational barriers facing women in medicine in her memoirs.

In 1892, Dr. Lilian Welsh came to Baltimore to enter private practice after studying medicine at the Women's Medical College of Pennsylvania and conducting research at the University of Zurich. But, the stimulating environment of the struggle to found The Johns Hopkins Medical School, the blossoming opportunities for women's undergraduate education, and the abject poverty and ill health suffered by many of Baltimore's women propelled Dr. Welsh into a life of service and education to improve the lives of women. She worked at the Johns Hopkins dispensary for poor women, served as professor of physiology and hygiene, director of physical education, and medical advisor to Goucher College, and held many other leadership roles within the health and suffrage movements.

Paramount in Dr. Welsh's mission was her belief that public health could solve public ills. She catalogues her work in public health: instruction, home health education, clean milk campaigns, the building of public baths, training of midwives, encouraging birth registration, tuberculosis control, and social services. Her belief that women should be involved in the advancement of society beyond

their families is reflected in an anecdote told by her colleague Dr. Gertrude Bussey: "I vividly remember the unholy glee with which she told of one poor freshman's answer to a question concerning her mother's occupation. In innocence or in bravado, the girl had replied, 'My mother's occupation is loving me.' I leave you to guess Dr. Welsh's retort."

In addition to describing her work in health and education, Dr. Welsh provides moving accounts of suffrage marches, explains the important role played by academic women in giving legitimacy to the movement, and compares the suffrage marches to those held fifty years before at the Women's Medical College of Pennsylvania for the right to practice in local clinics.

Dr. Welsh was highly conscious of her generation's turning point in medicine, education, and politics, and her comments seem as much directed to us and the future role we must play as they were to her Goucher students, whom she envisioned leading society in the future. As Dr. Welsh was deeply involved in many Baltimore institutions, the reader will delight upon seeing references to Johns Hopkins, the Lyric, and the Bryn Mawr School, among others. And finally, for every woman doctor this is an encouraging lesson that a little luck, a strong wit, and a "peppery tongue" will get you a long way.

ALSO OF INTEREST

► *A Tribute to Lilian Welsh.* Baltimore: Goucher College; 1938. 42 pages. Available at The Johns Hopkins Welch Medical Library.

KATE TULENKO, M.PHIL.,
AND ANNA BOWEN, M.P.H.

Ms. Tulenko is a fourth year M.D./M.P.H. student at Johns Hopkins University and Ms. Bowen is a fourth year medical student at the University of Wisconsin. ■

The CMERC Update, now in its second year, informs all Med Chi accredited CME sponsors about the activities of the Continuing Medical Education Review Committee (CMERC). The CMERC has received feedback (both negative and positive) from our accredited sponsors this past year. This exchange of information has kept us all better informed.

Report on meeting of ACCME (Accreditation Council for Continuing Medical Education)

Deusdedit Jolbitado, M.D., Continuing Medical Education Review Committee (CMERC) chair, and Carol Doctrow, CMERC staff, attended the ACCME's summer meeting in Chicago, July 10-11, 1997.

ACCME members are appointed by seven parent organizations, and over the past few years, many new faces have appeared on the council. The council members, as well as ACCME staff, were friendly and made us feel welcome. They are receptive to new ideas and to input from their constituents, including state medical societies. They are also efficient and systematic in conducting their business.

They covered a lot during the committee meetings on Thursday and the full council meeting on Friday. A discussion by the Strategic Planning Implementation Committee on whether to establish a single accreditation period, and if so, what the length of that period should be, is one example of the issues covered. This issue was not resolved.

We had the opportunity to meet and speak with several members of the council and its committees, including Thomas Pester, M.D., chair of the Committee for Review and Recognition (CRR), which reviews and recognizes the CMERC as an accrediting body. We were encouraged by Dr. Pester, as well as by Dr. Kopelow, executive director of ACCME, and several ACCME staff members, to send a representative to the ACCME's state medical society meeting on September 5 and 6. As it has in the past, this meeting conflicts with Med Chi's semiannual meeting, precluding staff from attending. Nevertheless, we have arranged for Barbara Hulfish, M.D., to represent the CMERC at the meeting in Chicago.

From the ACCME — A new system for accreditation

The ACCME has accepted, as a working document, the recommendations of the Ad Hoc Committee established to "review and evaluate the ACCME system for accreditation."

The Ad Hoc Committee proposed a new system for accreditation, including revised essentials and standards. As elements of the new system are finalized, approved, and implemented, we will keep you informed.

New CME sponsor accredited in Maryland

The CMERC awarded a two-year provisional accreditation to Copper Ridge Education and Research Institute in Sykesville. Congratulations to their CME committee and administration.

Preparing applications and reports with care

The content of reaccreditation applications and interim reports are of primary importance to the CMERC. However, you should be aware that the manner in which a reaccreditation application or interim report is prepared also sends an important message to the CMERC. When an application or report is prepared and assembled in accordance with instructions and the presentation is clear and orderly, this indicates to the CMERC that your committee and staff devote time and care to your CME program. When an application or report is submitted with materials in jumbled order, with excessive and/or unnecessary materials, with materials that are not appropriately referenced, labeled, and tabbed, this suggests to the CMERC that the CME program itself may be handled in a sloppy or haphazard manner.

Additionally, when an application or report is carelessly prepared, the CMERC's staff must devote time to locating, sorting, reorganizing, labeling, and tabbing materials. This time could be better spent in other activities, such as communicating with individual sponsors in the most timely manner, communicating with all sponsors about issues of common interest, providing technical assistance to sponsors experiencing difficulties, etc. **In the future, applications and reports that are not prepared in accordance with instructions will be returned to the sponsor for reassembly before being reviewed.**

Needs assessment and evaluation using outcomes data

Essential #2, regarding needs assessment, specifies that a sponsor is required to "include data sources which go beyond the sponsor's own perception of need." Many sponsors fulfill this requirement through the use of needs assessment surveys and feedback on evaluation forms, which are both valuable sources of information about educational needs. Some sponsors have begun to use outcomes data from quality monitoring and utilization review functions at their institutions as a source of needs information. The ACCME and the CMERC encourage the use of concrete data as the basis for planning CME activities and further encourage the use of subsequent data as a means of evaluating a CME activity. While a direct correlation between the activity and the change in a quality indicator cannot always be established, the information about subsequent physician behaviors can contribute to the CME committee's assessment of the value of an activity.

For example, a hospital discovers through its Pharmacy and Medications Committee that antibiotic "X" is being used inappropriately. In thirty percent of the occasions where antibiotic X is prescribed, less expensive antibiotic Y could be expected to work equally well. Based on this data, the CME committee plans an educational activity on the appropriate use of antibiotic X. Three months after the activity, the Pharmacy and Medications Committee reports that inappropriate use of antibiotic X has been reduced by 50%. This data

suggests that the educational intervention impacted physician behavior in a desirable way. If the resources are available, this CME committee could examine the change in prescribing practices of individual physicians with respect to antibiotic X in an attempt to better correlate the relationship between physician behavior and the educational activity. Or, physicians could be surveyed about the perceived reason for the change in their prescribing practices.

This source of needs assessment and evaluation information is not available to all sponsors, but is available to many, especially hospitals. Try to think of creative ways that it can be used to enhance the quality and relevance of your institution's CME program.

DEUSDEDIT L. JOLBITADO, M.D.

Dr. Jolbitado is the chair of the Continuing Medical Education Review Committee (CMERC) of the Medical and Chirurgical Faculty of Maryland. ■

The purpose of this newsletter is to inform CME chairpersons, CME committee members, and all interested physicians about the activities of Med Chi's Continuing Medical Education Review Committee (CMERC) and about the rules and procedures that affect the implementation of CME programs in Maryland. When appropriate, news from the ACCME (Accreditation Council for Continuing Medical Education) is included.

Med Chi Bicentennial Celebrations

Med Chi has already begun planning celebration activities for its bicentennial in 1999.

If you have ideas or suggestions, please call Margaret Burri at

410-539-0872 or 1-800-492-1056.



EPIDEMIOLOGY AND DISEASE CONTROL PROGRAM

201 West Preston Street, Baltimore, Maryland 21201 (410) 767-6700

September, 1997

Prevention and Control of Influenza

Introduction

Public health interest in influenza derives from the rapidity with which influenza epidemics evolve and the associated morbidity and mortality. This article contains a brief description of influenza virus, methods for prevention, a summary of influenza activity in Maryland for the 1996-97 influenza season, and information about influenza vaccine in the form of an adaptation of the Centers for Disease Control and Prevention's (CDC) Influenza Vaccine Information Statement (VIS). Copies of the full VIS can be obtained from the Maryland Department of Health and Mental Hygiene (DHMH) Center for Immunization at (410) 767-6679. The complete recommendations of the Advisory Committee on Immunization Practices (ACIP) for the prevention and control of influenza can be found in the April 25, 1997 issue of CDC's MMWR (Morbidity and Mortality Weekly Report), Volume 46, No. RR-9. That document can be downloaded directly from CDC's internet homepage (www.cdc.gov) or accessed through links to the CDC on the DHMH homepage (www.charm.net/~epi1).

Influenza virus

Three types of influenza virus are recognized: A, B, and C. Both influenza A and B have been associated with widespread epidemics. Type C has been associated only

with sporadic cases and localized outbreaks. Frequent viral mutation that results in the emergence of new antigenic variants necessitates annual reformulation and re-administration of influenza vaccine.

Prevention with influenza vaccine

Vaccinating persons at high risk before the influenza season (October through May) *each year* is the most effective measure for reducing the impact of influenza. Each year's influenza vaccine contains three inactivated virus strains (usually two type A and one type B) that represent the viruses most likely to circulate in the U.S. during the upcoming winter. The effectiveness of influenza vaccine varies depending on the age and immunocompetence of the vaccine recipient. When a good match exists between the vaccine and circulating strains of virus, influenza vaccine has been shown to prevent illness in 70%-90% of healthy persons aged <65 years. Influenza vaccine has also been shown to be 50%-60% effective in preventing severe illness, secondary complications, and death among elderly persons in nursing homes.

Prevention with antiviral drugs

Chemoprophylaxis is not a substitute for vaccination. The use of amantadine or rimantadine should be considered as an alternative strategy for certain individuals and

groups at high risk for severe illness and complications if infected with influenza A. These agents are not effective for influenza B. See the ACIP recommendations for more complete guidance. The antiviral agents may also be used for therapy of influenza A infections and outbreak control in institutions.

Surveillance

Although individual cases of influenza are not reportable in Maryland, outbreak reporting is mandated by state regulation. DHMH maintains guidelines for case identification, reporting, and management of influenza or influenza-like illness (ILI) outbreaks in long-term care facilities (LTCFs) such as nursing homes. Influenza activity in Maryland is tracked by 1) voluntary laboratory reporting of sporadic cases, 2) reports of influenza (and influenza-like illness) outbreaks, and 3) reporting of excess school absenteeism. Laboratory confirmed influenza is defined as: a) culture of influenza A or B virus, b) direct detection of influenza A viral antigen in a respiratory specimen by enzyme immunoabsorbant assay (EIA), or c) a four-fold rise in serum antibody titer. An outbreak of influenza or ILI in a LTCF is defined as two or more clinically defined cases or one laboratory confirmed case of influenza within a 10 day period. Excess school absenteeism in county schools is defined as absence $\geq 10\%$ with a predominance of absence due to respiratory illness.

1996-97 cases

Maryland had a large increase in the reported cases of influenza during the 1996-97 season in comparison to the 1995-96 season [299 reports (246 type A, 53 type B) compared to 58 influenza isolates (56 type A, 2 type B)]. Most of the cases (78%) were confirmed by culture, and 22% of the cases were confirmed by EIA alone. Eight of Maryland's isolates were sent to the CDC's laboratory and all eight were characterized as A/Wuhan/359/95-like (H3N2); that is the same as the H3N2 component of the 1996-97 vaccine.

Among the 275 reported cases in persons with known age, 112 (42%) were elderly (>65 years of age). Females accounted for 59% of

cases of known sex. The peak incidence of illness as judged by specimen collection date occurred in December [203 (73%) of 277 specimens with known date].

1996-97 outbreaks

During the 1996-97 influenza season, there were 83 reported outbreaks of influenza of which 24 (29%) were laboratory confirmed influenza; the remaining 59 (61%) outbreaks were classified as ILI. Of the 24 laboratory confirmed outbreaks in Maryland, 23 (96%) were influenza type A; one was type B. There were 1,662 cases associated with the 83 outbreaks. (Laboratory confirmed outbreak *cases* are included in the case reports above; epidemiologically linked cases are not.) The outbreaks resulted in 65 hospitalizations and 21 deaths. During the 1995-96 season, 19 outbreaks were reported with 416 cases, 16 hospitalizations, and three deaths.

Among the 83 outbreaks of the 1996-97 season, the highest number of outbreaks [21 (25%)] occurred in Montgomery County, followed by 16 (19%) in Baltimore County, and 9 (11%) in Washington County. Outbreak activity occurred from October 1996 to April 1997 with the peak occurring in December. Most outbreaks (81, 98%) occurred in LTCFs; two (2%) occurred in correctional facilities.

1996-97 Excess School Absenteeism

There was a total of 73 reports of excess absenteeism compared to an average of 50 reports over the previous five years. Absenteeism peaked in December with 71 (97%) reports.

INFLUENZA VACCINE

WHAT YOU NEED TO KNOW

1997-98

1. Why get vaccinated?

Influenza (sometimes called flu) is a serious disease. Here are a few important facts:

It spreads when influenza viruses pass from an infected person to the nose or throat of others.

Influenza can cause:

- fever • cough • chills • sore throat
- headache • muscle aches

Influenza can make people of any age ill. Although most people are ill for only a few days, some have a much more serious illness and may need to be hospitalized. Thousands of people die each year from influenza-related illnesses. Most deaths caused by influenza are in elderly people.

2. Influenza vaccine

The viruses that cause influenza change often. Each year a new influenza vaccine is made using viruses that are thought to be most likely to come to the United States, or ones very similar to them. This year the vaccine contains these viruses:

A/Johannesburg/82/96 (H1N1)
A/Nanchang/933/95 (H3N2)
B/Harbin/07/94

3. Who should get influenza vaccine?

Group #1. People who are at risk for getting a serious case of influenza or a complication should get the vaccine. This includes:

- All people 65 years of age or older.
- Residents of long term care facilities housing persons of any age with chronic medical conditions.
- Any child or adult, including pregnant women, who has a serious long-term health problem with: - heart disease - lung disease - anemia - kidney disease - metabolic disease

such as diabetes - asthma

AND in the past year had to: - see a doctor regularly, or - be admitted to a hospital

- People who are less able to fight infections because of: - a disease they were born with - infection with Human Immunodeficiency Virus (HIV), the virus that causes AIDS - treatment with drugs such as long-term steroids - cancer treatment with x-rays or drugs
- Children and teenagers 6 months to 18 years of age on long-term aspirin treatment, who, if they catch influenza, could develop Reye's syndrome which causes coma, liver damage, and death.
- Women who will be more than 3 months pregnant during the influenza season.

Group #2. Anyone who has close contact with people who are at risk for getting a serious case of influenza. This includes:

- Anyone-including children-who live with people in high risk groups (Group #1 above)
- Health care workers (doctors, nurses, hospital and medical office staff)
- Personnel of nursing homes or chronic care facilities
- People who provide home-care to high-risk persons, such as visiting nurses and volunteers

Group #3. In addition, an influenza shot may be given to:

- Persons who provide important community services
- People in schools and colleges, to prevent outbreaks
- People going to the tropics any time of year or to countries south of the equator between April and September
- Anyone who wants to reduce their chance of catching influenza

4. When should I get influenza vaccine?

People who need the vaccine should get it every year.

The vaccine begins to protect you after 1 to 2

weeks and protection may last up to one year. Influenza is most common in the U.S. from December to April, so it is best to get the vaccine between September and mid-November. People 9 years and older need one shot each influenza season. Children less than 9 years old may need a second shot after one month. Influenza vaccine can be given at the same time as any other vaccines, including pneumococcal vaccine. It should be given in a different limb.

5. Can I get the influenza even though I get the vaccine this year?

Because the viruses change often, they may not always be covered by the vaccine. But people who do get influenza after getting the vaccine often have a milder case than those who did not get vaccinated.

Also, other viruses cause disease that seem like influenza, and the influenza vaccine does not protect against these other viral infections.

6. What are the risks from influenza vaccine?

As with any medicine, there are very small risks that serious problems, even death, could occur after taking the vaccine. The risks from the vaccine **are much smaller** than the risks from the disease if people stopped using vaccine. Almost all people who get influenza vaccine have no serious problems from it.

Children less than 13 years old should be given only split virus vaccine to reduce chances of side effects. Split-virus vaccine can also be used by adults.

If mild or moderate problems occur, they usually start soon after the vaccination and can last up to 1-2 days. These may include:

- soreness, redness, or swelling where the shot was given
- fever
- aches

In 1976, swine flu vaccine was linked to a severe paralytic illness called Guillain-Barre' Syndrome (GBS), from which about half its victims fully recover. Since then, other influenza vaccines have not been clearly linked to GBS. However,

in 5 of 6 years studied since 1976, there may have been a small chance that getting GBS was linked to influenza vaccine. The chance of GBS after influenza vaccine is far less than the chance of getting severe influenza that could be prevented by the vaccine.

The viruses in the vaccine are killed, so you cannot get influenza from the vaccine.

7. Tell your doctor or nurse if you:

- have a serious allergy to eggs
- ever had a serious allergic reaction or other problem after getting influenza vaccine
- were ever paralyzed by Guillain-Barre' Syndrome
- now have a moderate or severe illness

8. What if there is a problem after vaccination?

What should I look for?

A severe allergic reaction could include hives, difficulty breathing, or shock.

What should I do if it is a serious problem?

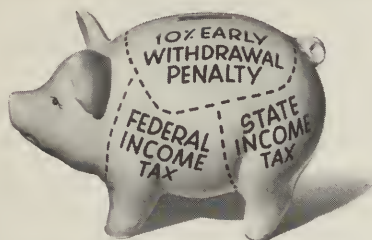
- Call a doctor or get the person to a doctor right away
- Tell your doctor what happened, the date and time it happened, and when the vaccination was given.
- Ask your doctor, nurse, or health department to file a Vaccine Adverse Event Reporting System (VAERS) form. Or call VAERS yourself at 1-800-822-7967.

9. How can I get more information?

- Ask your doctor or nurse. She/he can give you the vaccine package insert or suggest other sources of information.
- Call your local or state health department.
- Contact the Centers for Disease Control and Prevention (CDC)
 - Call 1-800-232-7468 (English) or 1-800-232-0233 (Spanish)
 - Visit the CDC website at <http://www.cdc.gov/nip>

Adapted from Centers for Disease Control and Prevention, Influenza - 7/1/97 Vaccine Information Statement

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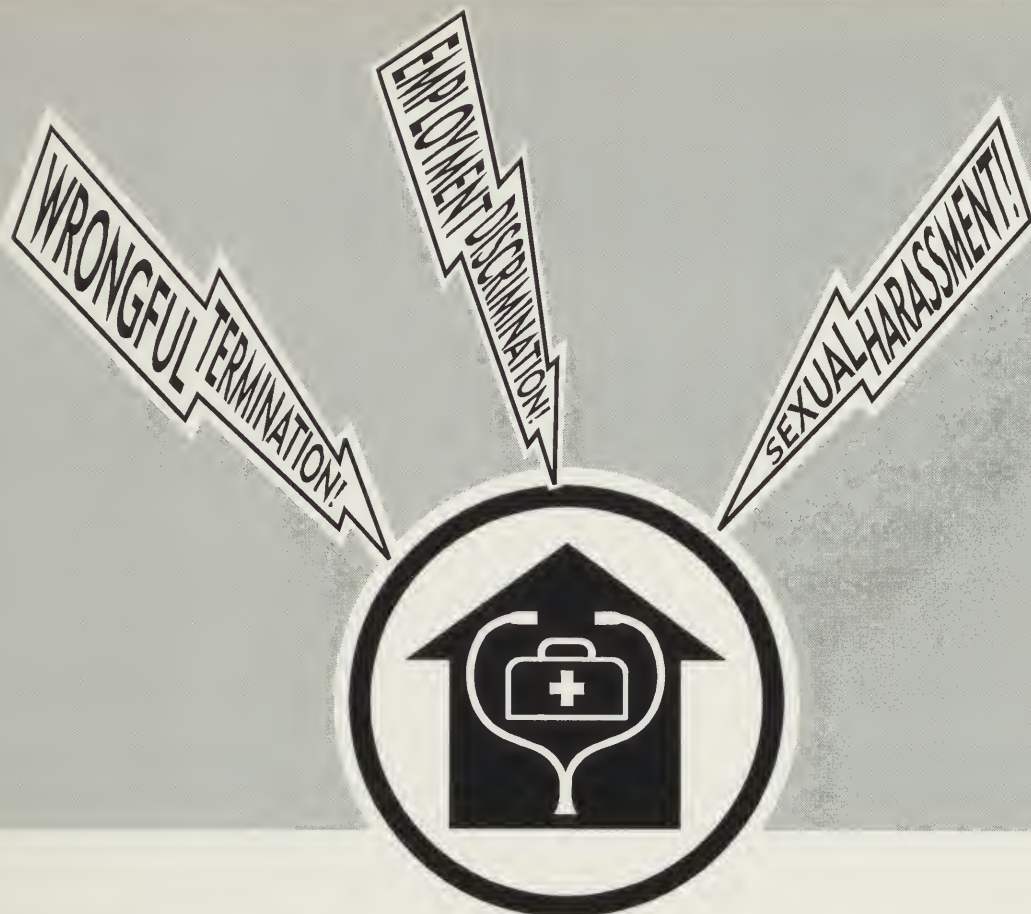
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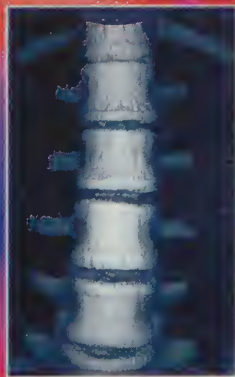
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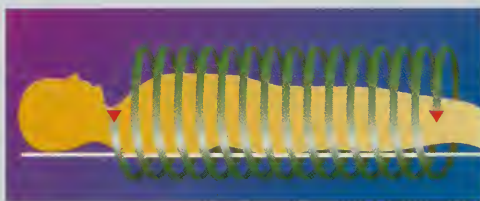
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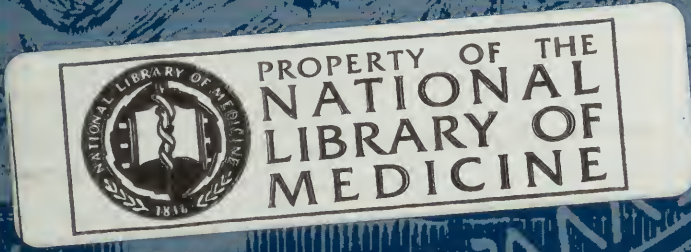
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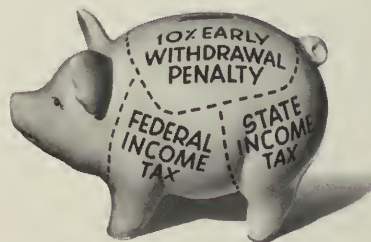
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ERRATUM

On p. 424 of the September 1997 issue of the *MMJ*, Dr. Zorayda M. Lee Llacer's biosketch was incorrect. It should have read:

BIOSKETCH

- Medical degree: *University of Santo Tomas in Manila, Philippines*
- Internship: *Prince George's Medical Center, Cheverly, MD*
- Residency: *Anesthesia, Georgetown University Medical Center, Washington, DC*
- Prior positions: *Assistant professor, department of anesthesia, Georgetown University Medical School; Director respiratory therapy department, Georgetown Medical Center*
- Present positions: *Medical director, medical surgical intensive care unit, Doctors Community Hospital, Lanham, MD; Secretary, Prince George's County Medical Society; Member, House of Delegates, Med Chi*

The *MMJ* regrets the error.

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Report on Hopkins' meeting: Nothing is sacred

I recently attended the biennial meeting of the Johns Hopkins Medical and Surgical Association. I had hoped to meet some of my former classmates and colleagues there, as well as learn a bit about recent advances in medicine at my alma mater.

As it turned out, I accomplished neither of these goals. But I did verify that Hopkins, along with everything else in medicine, has joined the business community and turned its attention to financial as well as scientific affairs. At the plenary session the two speakers were the new dean and chief executive officer of "Johns Hopkins Medicine," Edward Miller, M.D., and the president and chief executive officer of Johns Hopkins Health System and Hospital, Ronald Peterson.

They essentially had nothing to say about what was new in the laboratories and on the wards at Hopkins and a great deal to say on the subject of medical economics. There was the mention of marketing the Johns Hopkins trademark and negotiating with various hospitals and physician groups in the area. Insurance companies drew a lot of discussion since they have the patients and the money to pay the bills.

Mr. Peterson made it clear that even the famed Hopkins name would not attract payers to permit their clients (previously referred to as patients) to come to the hospital unless some way could be found to reduce the additional costs resulting from research and residency programs. I believe that these programs, which are the essential backbone of continued excellence and progress in medical care, raise the hospital rates about 30% above those of non-academic institutions; and these charges must be cut by one-third or more to attract payment by that comptroller of medical money — the insurance industry.

It was not entirely clear to me (and I admit that my understanding of such matters is limited) if the decrease in hospital charges was to come from reduced teaching and re-

search costs, new sources of funds, or both. It was mentioned that Hopkins recognized that its reputation and responsibility to maintain high standards for its students, practitioners, scholars, and patients required continuation of its present academic as well as more entrepreneurial activities.

Dr. Miller, who appears a gregarious, homespunky kind of chief executive officer, has been serving as interim dean since Dr. Michael Johns accepted a post at Emory about a year ago. He seems to have had long-standing relationships with many of the Hopkins faculty that antedated his job as chief of anesthesia at Hopkins that began in 1994. He is said to have a talent for mediating disputes among faculty members, as well as a respectable record of academic and anesthesiology achievement.

Dr. Miller served as credible co-star with Mr. Peterson at the plenary session, where his affable and informal manner contrasted with the concerned and cautious attitude of the businessman reporting that profits may not be as good as expected in the near future. I thought there were definite indications that compromise of some type, either in programs or alliances, might be necessary for financial survival.

The latest issue of *Hopkins Medical News* verified the more optimistic approach of Dr. Miller, who hopes that such innovations as licensing of technology, partnering risk with faculty members, capitalizing on methods and protocols, and soliciting further National Institutes of Health (NIH) funding may help maintain the Hopkins' tradition. Drug companies as a source of revenue was another of his ideas.

From the standpoint of an aging practitioner, who through 40 years of clinical practice has been able to avoid more than just an occasional interest in financial matters, these ideas are troubling. The idea of licensing medical methods, for example, brings con-

LETTERS TO THE EDITOR

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cern that treatments may be kept secret and sold to the highest bidder, that the rift between care of the poor and that of the rich may grow wider, and that developing countries will not be able to afford the knowledge to help their impoverished citizens.

Sharing risk is a concept that carries with it the stigma of physicians reducing referrals to specialists and not ordering tests because their income will be less. I associate it with the capitation system that pays the physician a certain fixed amount per enrollee and makes us dread the sick patient and try to keep him out of the hospital for fear he will become a personal financial burden. Of course, there are those who, with some justification, feel a fee-for-service payment system leads to excessive therapeutic interventions.

I worry also about too much dependence on NIH funding, with the present conservative political philosophy engulfing the country. For example, there has been much discussion about denying money for fetal research lately. Even closer to home is worry that the reasonable Hopkins' approach to family planning and more recently gun control will result in reductions in federal funding by fundamentalists and militia-minded legislators.

I have a personal prejudice against research funded by pharmaceutical companies, which may not be entirely justified, but makes me suspicious of many results. The recent publicity over the squelching of evidence that a generic thyroid preparation was equal to Synthroid by a drug company may be evidence of an institutional problem. Even more troubling are studies which show that taking a drug such as estrogen is associated with longevity and publicizing them in a way that suggests an unproved cause and effect relationship. Such results are usually followed by editorials lamenting the failure of practicing clinicians to act expeditiously in prescribing for their patients based on such inadequate evidence. The recent call for anticoagulants for so many elderly patients with atrial fibrillation suffering from concomitant diseases and taking multiple other medicines may be another case in point.

Although I certainly hope that Dr. Miller and Mr. Peterson can lead Hopkins into the new millennium with its tradition of excellence intact, I tend to be leery about bringing in businessmen to run our medical projects with "businesslike concepts." Mercenary attitudes seem to

breed selfishness and preoccupation with short-term goals, which may be incompatible with the requirements of proper medical care, instruction, and research. Perhaps the insurance companies' stockholders and chief executive officers will be forced by legislation to give up some of those obscene profits that I have been reading about and reduce the need for risks for patients and physicians.

Perhaps, also, some physicians who may have gotten the idea that third-party payments were a limitless source of increasing "usual and customary fees" will learn that they have slain the goose. When a procedure, whether it be cataract surgery, endoscopy, or measuring cholesterol, becomes easier and less time consuming to perform, it might be more appropriate to reduce the charges rather than attempt to increase the indications. We do not need to be reminded that Medicare discovered this before we did.

Next year, I shall know a little better what to expect at the Medical and Surgical Association meeting when the new "entrepreneurial road" of Dr. Miller has had a chance to begin to reap what is sown.

NELSON G. GOODMAN, M.D.

Dr. Goodman practices internal medicine in Bowie, Maryland. ■



Physician seeks information on disability coverage

I am a board certified obstetrician and an advanced laparoscopist by training. Due to an accident, I became disabled about two years ago. The insurance coverage that was supposed to cover me for disability was paid for a period of time. The insurance company has now taken the position that I am not disabled because I am able to participate in certain limited activities with my children.

I have severe inoperable residual symptoms from a slipped disc, which has been explored surgically. In addition, I have lost much of the function of my dominant hand due to a residual ulnar nerve palsy complicated by reflex sympathetic dystrophy. I am obviously unable to practice my specialty because of the combination of the loss of function and sensation of my dominant hand as well as my residual back symptoms.

I would be interested to know about other physicians' experiences with insurance companies related to disability coverage, most specifically with their insurance carrier.

Please communicate with me. I look forward to hearing from you.

SHEILA E. BUCHBINDER, M.D.

P.O. Box 21, Ringoes, NJ 08551 ■

Allan C. Gelber, M.D., M.P.H., is lead author of "Gout and Risk for Subsequent Coronary Heart Disease," published in the July 14 issue of *Archives of Internal Medicine* (1997;157:1436-1440). The results of this study did not indicate targeting gout identification in the primary prevention of coronary heart disease. Dr. Gelber is from the departments of medicine and epidemiology, Johns Hopkins Medical Institutions. Other authors include **Michael J. Klag, M.D., M.P.H.**, also from Johns Hopkins, and **Marc C. Hochberg, M.D., M.P.H.**, from the departments of medicine, epidemiology, and preventive medicine, University of Maryland School of Medicine, and the Geriatric Research Education and Clinical Center, Veterans Affairs Medical Center, Baltimore.

Una D. McCann, M.D., is among the authors of a review article indicating that use of dexfenfluramine and fenfluramine result in a reduction in brain serotonin in animals. In the article, published in the August 27 issue of *JAMA* (1997;278:666-672), the authors wrote, "loss of serotonin axonal markers after fenfluramines is evident weeks, months, and in one primate study, as long as one year after drug discontinuation." Although it is not known whether the drugs will exact the same effects on humans, the authors indicate that an estimated 50 million persons have taken fenfluramines. Dr. McCann is from the National Institute of Mental Health, Bethesda.

Ansgar M. Brambrink, M.D., Christoph Lehmann, M.D., Jeffrey Rothstein, M.D., Ph.D., and colleagues wrote "Hypoxia-Ischemia Causes Abnormalities in Glutamate Transporters and Death of Astroglia and Neurons in Newborn Striatum," in which they "tested the hypothesis that damage to astrocytes and loss of glutamate transporters accompany striatal neurodegeneration after hy-

poxia-ischemia" (*Ann Neurol* 1997;42:335-348). Drs. Brambrink, Lehmann, and Rothstein, and colleagues are from the Johns Hopkins University School of Medicine, where Dr. Brambrink is in the department of anesthesiology/critical care medicine, Dr. Lehmann is in the department of pediatrics, and Dr. Rothstein is in the department of neurology.

Ronald D. Berger, M.D., Ph.D., is lead author of an article on the new electrical abnormality found in the heartbeat of heart failure patients, published in the September 2, 1997, issue of the journal *Circulation*. The authors of this study found that some heart failure patients have an electrical abnormality that prevents the heart from recovering normally after each beat. The researchers compared 83 patients with dilated cardiomyopathy with 60 control subjects who had no evidence of heart disease. The research was supported by two grants from the National Heart, Lung and Blood Institute and from Johns Hopkins. Dr. Berger is an assistant professor of medicine at Johns Hopkins. Other study authors were **Edward K. Kasper, M.D., Kenneth L. Baughman, M.D., Eduardo Marban, M.D., Ph.D., Hugh Calkins, M.D., and Gordon F. Tomaselli, M.D.**

Researchers reported the identification of the first known genetic mutation that causes familial colorectal cancer in the September 1, 1997, issue of *Nature Genetics*. The research team studied blood and tissue samples of 211 Ashkenazi Jewish colon cancer patients and found that one in six of those who developed cancer before age 66, and one in eight of those who developed cancer at any age, had the mutation. Study authors from the Johns Hopkins Medical Institutions included **Frank Giardiello, M.D., Stephen Gruber, M.D., Ph.D., Stanley Hamilton, M.D.**, and colleagues. Dr. Giardiello is director of the Hopkins Hereditary Colorectal Cancer Clinic and Registry.

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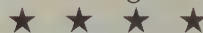
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Postmortem cesarean section with infant survival: a case report of an HIV-infected patient

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Dr. Esposito is from Sinai Hospital of Baltimore, Mr. DeLony is from the University of Baltimore School of Law, and Dr. Goldstein is from the Washington Hospital Center and is associate professor of OB/GYN, Johns Hopkins University.

ABSTRACT: *Postmortem cesarean sections are rare events, but modern technology is forcing society to explore the definition of viability and the legal rights of both mother and fetus that ultimately will affect the frequency and use of this rare operation. Since the Human Immunodeficiency Virus (HIV) prevalence rate and the number of critically ill obstetrical patients with Acquired Immunodeficiency Syndrome (AIDS) continues to rise, it is reasonable to assume more patients and physicians may be confronted by issues concerning fetal and maternal rights and other considerations in perimortem delivery.*

A 33-year-old woman, at 27 weeks' gestation, with fulminant AIDS was admitted to the intensive care unit (ICU) in respiratory distress. As her condition deteriorated the complex problem of caring for both mother and fetus emerged. A patient advisory panel explored the issues with the patient, her family, and her health care team. Eventually a peri/postmortem cesarean section was performed on the mother when she suffered an acute fatal cardiorespiratory arrest.

With the prevalence of AIDS increasing and with most women not being tested prior to pregnancy, many obstetricians will be confronted with new medical and legal challenges. Establishing comprehensive medical management for the critically ill obstetrical patient and understanding the legal rights of both patients (mother and fetus) will help avoid conflicts and potentially improve survival.

Introduction

"Postmortem cesarean section remains, at best, an obstetric calamity. More often than not, the delivery of a stillborn infant or badly compromised child who does not survive compounds the tragedy of the mother's death."¹

Postmortem cesarean section is not a new concept and can be traced through ancient history, mythology, and folklore.¹⁻³ Yet each century, physicians have had to face changing criteria for its application and approach.

Over the last decade, advances in the medical technology of both obstetrics and neonatology have enhanced the potential for infant survival. In light of these changes, Katz et al., suggested a re-examination of the practice and use of postmortem cesarean sections.³ He advocated that with prompt and appropriate management, a healthy infant can be saved by a postmortem or perimortem cesarean section. Under these circumstances, the possibility exists, therefore, that the rights of the fetus will increasingly conflict with the autonomy of the mother.⁴⁻⁶

As the twentieth century draws to a close, obstetricians must prepare for the consequences of the widespread Acquired Immunodeficiency Syndrome (AIDS) epidemic. AIDS has quickly become the fourth leading cause of death in the United States for women of reproductive age.^{7,8} It has been estimated that annually, in the United States, about 6 000 pregnant women will deliver babies with the Human Immunodeficiency Virus (HIV).⁷ Obstetricians, therefore will likely be caring for an increasing number of women with this disease. The following case report of a patient with AIDS, whose baby was delivered via postmortem cesarean section, raises several medical and legal issues obstetricians will be forced to confront.

Case report

A 33-year-old woman, para 3, gravida 2, at 27 weeks' gestation, presented to the emergency room with a two-week history of fever, nonproductive cough, right pleuritic chest pain, and shortness of breath. She also reported a four-day history of myalgias, anorexia, vomiting, and drenching night sweats. The patient initially sought prenatal care at 21 weeks' gestation but had no further prenatal care. The serology drawn at the first prenatal visit was significant for a positive rapid plasma reagin (RPR) (1:128), a reactive fluorescent treponemal antibody (FTA), and a positive enzyme linked immunoabsorbent assay (ELISA) and Western Blot for HIV.

The medical history was significant for a blood transfusion requiring four units of packed red blood cells for an unspecified anemia seven years prior to admission and acute pyelonephritis the year of admission. The social history was

positive for parenteral cocaine abuse and a two pack per day history of cigarette smoking. She denied alcohol use and admitted to only one sexual partner.

Upon physical examination, the patient was an ill-appearing female with a violent, nonproductive cough and shortness of breath. Her blood pressure was 120/70 mm Hg, her pulse was 120, her respiratory rate was 38, her temperature 37.9°C, and the fetal heart rate was 148. The examination was unremarkable except for chest auscultation with bronchial breathing and scattered rales. The uterine fundal height was 26 cm. On pelvic examination the cervix was long and closed and uterine size was consistent with dates. An arterial blood gas (ABG) at room air was pH, 7.39; PCO₂, 31; PO₂, 66; O₂ Sat, 93%. The white blood cell count was 7600 and the hematocrit 30%. The chest x-ray demonstrated diffuse bilateral interstitial infiltrates with a right perihilar mass. The echocardiogram (EKG) indicated sinus tachycardia but was otherwise within normal limits. The patient developed worsening respiratory distress and was unable to maintain adequate oxygen saturation despite 40% oxygen supplementation. She was therefore electively intubated and admitted to the intensive care unit (ICU). The working diagnosis was that of pneumonia, most likely secondary to *Pneumocystis carinii*. Consultation to assist in the management included the departments of obstetrics, pulmonary medicine, infectious disease, and social work.

During the first several days of admission, she remained ventilator dependent but stable. A fiber-optic bronchoscopy was done with bronchoalveolar lavage and cultures were obtained. Broad-spectrum antibiotic therapy was initiated to treat syphilis and to empirically treat the multiple potential differential etiologies of pneumonia. Blood cultures previously drawn in the emergency room grew *Cryptococcus neoformans*. The diagnosis of AIDS was then confirmed when a positive silver stain for *Pneumocystis carinii* was obtained.

By the end of the first week of admission, the patient's condition slowly deteriorated. Because of the patient's critical status, the family was asked for their assistance in developing a management plan, especially for the fetus, since maternal death seemed inevitable. A patient care advisory committee meeting was convened to help the family address the issue from ethical, legal, and medical perspectives. The committee consisted of physicians, social workers, and nurses. The family understood the patient's condition and her prognosis. They understood the potential consequences to the neonate posed by prematurity, maternal infectious disease, and postmortem cesarean section. They were informed regularly with respect to maternal and fetal status, and had access to a variety of professional sources of support and information. Together with the mother, they agreed to cesarean

section and to infant care support by the family after the infant's discharge from the hospital.

On the twentieth hospital day, the ICU notified the obstetrical staff of the patient's impending demise. At 2:00 p.m. she became hypotensive and experienced a drop in systolic blood pressure to the 60s and heart rate to the 40s. An EKG noted an acute inferior wall myocardial infarction. Despite the use of heroic chemical and ventilator support, the patient developed cardiac asystole. Chest compression was started, and at 2:16 p.m., an emergency cesarean section was performed in the ICU. A preterm appropriate for gestational age (AGA) male infant with Apgars scores of 2 and 6 was delivered and handed over to the waiting neonatologist. A 90% abruption of the placenta was noted at the time of the delivery. The patient continued without blood pressure or pulse, cardiopulmonary resuscitation was discontinued, and she was pronounced dead at 2:35 p.m. The infant was intubated in the ICU and taken to the neonatal intensive care unit. All bacterial cultures were initially negative, as was the RPR and ELISA. The infant was ultimately discharged to a long-term care facility secondary to chronic respiratory complications.

Discussion

Although the number of peri/postmortem cesarean sections have increased in modern times, it is still a relatively rare event. Because of the advancing epidemic of AIDS it may tragically become more prevalent, especially since most 18- to 44-year-old women are not being tested prior to pregnancy.⁹

Previously, most peri/postmortem cesarean sections resulted from an acute maternal event (i.e. maternal trauma or illness).² Because of the nature of the emergency, little or no time was available for discussion of the mother's wishes regarding her care of the fetus. Conventional medical wisdom dictated that in the face of catastrophic maternal events, physicians should perform an immediate perimortem cesarean section.³ Physicians acted on the presumption that the procedure could benefit both mother and fetus, without regard to potential legal liability.

Recently, because of the ability to rescue very premature infants and because of ambiguity in mothers' rights versus fetal rights, planned "emergency" cesarean sections have increased in women who are dying or arguably already dead. For these situations, a personalized obstetrical management plan for each chronically ill patient must be developed.¹⁰ A physician offering antepartum care in such cases needs to explore the following topics: 1) defining the patient's wishes for treatment of her disease (and the consequences of her treatment on her and her fetus), 2) establishing appropriate informed consent, 3) preparing the patient and family for the untoward event, and 4) informing the patient of her rights and

the medical and legal issues that a terminally ill obstetrical patient faces.

While developing a medical management plan, the patient must consider several issues, including the effects of prematurity and chronic medical illness on the infant, and the potential burden such a child may have on those who assume the responsibility for raising the child. Minimum informed consent on these issues requires an explanation of the potential chronic illnesses the baby may suffer, and of infant survival rates.

The patient must be managed in a facility skilled in high-risk maternal and neonatal patients. At 25 to 28 weeks' gestation, a dramatic change in infant morbidity and mortality occurs, with the chances of survival increasing from 30% to 75% and with a corresponding decrease in morbidity.¹⁰ This contrast in survival and morbidity should play a factor in the decision of whether and when to do a perimortem or postmortem cesarean section.¹¹

The best chance of neonatal survival of peri/postmortem cesarean section occurs when the time between maternal death and delivery is reduced as much as possible.³ One suggested guideline is the "Four Minute Rule"—the amount of time allowable between maternal death and delivery of the infant before major morbidity occurs. This rule derives from studies of the effects of experimental anoxia on the mother and fetus in primates that demonstrated that most fetal survivors are delivered within four minutes of the mother's demise.¹² Rare cases have documented survival when postmortem cesarean sections were performed after the four minute limit.¹³ Thus, physicians may be justified in performing a delayed postmortem cesarean section if fetal heart tones are still auscultated. Decreasing the time to delivery requires proper preparation, including 1) prior documentation of the patient's wishes regarding intervention on behalf of the fetus with family inclusion where possible, or, in the case of the patient's inability to make her wishes known, the recommendation of an ethics committee on how to proceed; 2) an established hospital policy on procedure in the absence of such a recommendation; 3) an established emergency notification protocol of the hospital staff, including obstetrical and neonatal personnel in the event of impending maternal demise; and 4) readily available equipment for delivery and resuscitation of the infant at the maternal bedside.

Legal issues. When making decisions regarding peri/postmortem cesarean sections, physicians must the rights of their two patients, the mother and the fetus, and understand the government's interests in maternal health and in the potential life of the fetus. The judicial balancing of these two separate, and at times, conflicting interests can affect the dynamics of the doctor/patient relationship.

The U. S. Supreme Court has recognized a woman's privacy interest, as well as the state's right to protect potential life. *Roe v. Wade* established the principle that viability marks the earliest point at which the state's interest in fetal life is constitutionally adequate to justify legislative ban on nontherapeutic abortions.¹⁴ Although *Roe* is generally regarded as "the abortion ruling," *Roe* actually also established a trimester framework that allowed the state to intervene on behalf of the fetus to protect potential human life. *Roe* allows the mother in the first trimester to terminate her pregnancy before viability without undue interference from the state, allows the state in the second trimester to intervene in the interest of the mother's health, and allows the state in the third trimester to intervene on behalf of the fetus to protect the state's interest in potential human life.¹³ The trimester framework may, however, no longer be favored by the Supreme Court, as evidenced in *Webster v. Reproductive Health Services*.¹⁵ *Webster* can be interpreted as creating a legal presumption of viability at 24 weeks, which is earlier than the point of viability discussed in *Roe*. The *Webster* Court also upheld the constitutionality of the Missouri state policy declaring that the state's interest in potential human life arises when the fetus comes into existence (i.e., at the time of conception).

When the two separate interests conflict, some physicians have unfortunately resorted to obtaining judicially enforced orders to provide what they consider necessary to care for the fetus. Thus legal institutions may become involved in resolving the medical/legal question as to whom the physician is ultimately responsible. The American College of Obstetrics and Gynecology (ACOG) has attempted to define the physicians' role in this complex legal environment. In 1987 and 1989, ACOG stated that while every reasonable effort should be made to protect the fetus, the pregnant woman's autonomy should be respected and no other party, including the state, should override her autonomy.^{16,17} ACOG declared that the use of the courts has a destructive effect on women's autonomy and on the physician/patient relationship, and is almost never warranted.¹⁶

One way to avoid the tragedy of pitting the mother's rights against fetal rights is to address possible conflicts of interest before legal intervention is sought. Discussion of medical, legal, and ethical issues with the terminally ill patient and her family, early in the course of prenatal care, should be encouraged. Physicians should help the patient to clarify her wishes regarding her care and the care for the fetus. In recent years, many states have enacted "living will" statutes, whereby a terminally ill patient can execute a directive accepting or refusing medical procedures in the

event she is unable to communicate her wishes and death is imminent. A legally incompetent adult patient is generally held to have the same constitutional right to refuse treatment accorded to a competent adult patient. However, the legality of the advance directive may be questioned by anyone seeking to override the patient's wishes. In the case of a patient's directive refusing a perimortem or postmortem cesarean section, courts may find that the fetus' right to life outweighs the patient's liberty interest in avoiding unwanted medical treatment.⁶ Fetal rights issues are burgeoning in the law.⁴⁻⁶

While always calamitous, fortunately, maternal mortality and postmortem cesarean sections are rare obstetric occurrences. Only through timely, well-planned hospital policies and thoughtful doctor/patient communication can the rights of the mother be respected and the best possible outcomes for the fetus be achieved.

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Multimodality treatment in the management of esophageal cancer: neoadjuvant chemoradiotherapy followed by transhiatal esophagectomy

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and Armando Sardi, M.D., FACS

From the departments of surgical, medical, and radiation oncology, St. Agnes HealthCare, where Dr. Irwin is a medical student, Dr. Waterfield is chief medical oncology, Dr. Poussin-Rosillo is chief radiation oncology, and Dr. Sardi is chief surgical oncology.

ABSTRACT: *Esophageal cancer, although not one of the more common malignancies in the United States, remains a significant problem. Nearly as many patients as are diagnosed die in the same year, regardless of the treatment employed. Surgery is considered the mainstay of therapy. Esophagectomy with the use of the stomach as a substitute is preferred. Radical procedures have not proven more effective in extending survival. Because of the poor five-year survival rate, multimodality therapy with preoperative chemoradiotherapy (neoadjuvant therapy) followed by esophagectomy has shown encouraging results. Two illustrative cases are presented, one with adenocarcinoma and one with a squamous cell carcinoma, that were treated in this manner.*

Introduction

Esophageal cancer was responsible for 12 300 new cancer cases and 11 200 cancer deaths in the United States in 1996.¹ Cancer of the esophagus is most common in elderly men, with a male to female ratio of 3:1. It is more common in blacks than whites. Greater than 50% of patients present with advanced disease at diagnosis. The overall five-year survival rate is less than 10%.² In general, the treatment of esophageal cancer is considered to be palliative by most physicians.

Treatment modalities include surgical resection, chemotherapy, radiation therapy, and combinations of these modalities. Surgical resection alone provides excellent palliation of dysphagia and increased survival compared with the other forms of therapy, especially in patients with early stages of disease. Unfortunately, most patients present in stages III or IV. Radiation therapy alone in the treatment of esophageal cancer has also shown poor results, even at higher doses of radiation. Median survival after radiation therapy is about 12 months. The best results of radiation therapy alone were

reported by Pearson et al., in which a five-year survival rate of 17% was achieved.³ However, the universal experience with radiation therapy alone has been less effective. More than 8400 patients in 49 series demonstrated a five-year survival of only 6%.⁴ Radiotherapy alone is considered an effective palliative treatment of esophageal cancer. Chemotherapy as single treatment of esophageal cancer is considered investigational. Duration of response to single agent or combination chemotherapy is brief, lasting less than three months and palliation of symptoms is minimal. When used preoperatively (neoadjuvant therapy), reduction of tumor bulk occurs in 40% to 60% of patients, but histologically complete responses are infrequent (1% to 3%).⁵ At the present time, surgery remains the treatment of choice for resectable tumors, but the poor five-year survival rates have led to forms of multimodality therapy in search of better results.

This paper discusses the multimodality approach for the treatment of esophageal cancer. Two illustrative cases, one with adenocarcinoma and one with squamous cell carcinoma, who received neoadjuvant chemoradiotherapy followed by transhiatal esophagectomy are presented.

Case 1

A 61-year-old white man presented on October 16, 1994, with dysphagia and weight loss of two months' duration. Biopsy obtained by esophagogastroduodenoscopy (EGD) revealed a well-differentiated adenocarcinoma of the distal esophagus with marked dysplasia. Computed tomography (CT) scan revealed the tumor to be 2 cm below the carina extending to the esophagogastric junction with no apparent spread to the surrounding structures. He underwent preoperative chemoradiotherapy. A total dose of radiation over five weeks consisting of 4500 cGy in 25 fractions, 3600 cGy was delivered to the mediastinum and celiac axis and an additional 900 cGy was delivered to the esophagus only. Chemotherapy was administered on the first and fifth week of radiotherapy. It consisted of 300 mg/m² 5-fluorouracil and 60 mg of cisplatin by continuous IV infusion daily for five days, and three doses of VP-16 at 120 mg on days one, three, and five. Preoperative CT scan showed partial response. This was followed by transhiatal esophagectomy on January 10, 1995. The patient had an uneventful postoperative recovery. The intraoperative blood loss was 500 cc. No blood transfusion was required. He was discharged on the ninth postoperative day without complications. The resected specimen demonstrated residual disease. The specimen showed well-differentiated adenocarcinoma and the tumor invaded through the muscularis. One of five nodes was positive for malignancy, a pathologic stage III. The patient was asymptomatic with a 100% Karnofsky performance status until June 3, 1996, when he presented with left hemiparesis, worse in the left upper extremity. Magnetic resonance imaging (MRI) of the head revealed a single 2.5 x 3.0 cm lesion situated in

the right posterior parietal lobe under the motor strip for the left upper extremity, consistent with a solitary metastasis. Work-up for other metastases was negative and his symptoms improved with dexamethasone.

The patient underwent a right frontoparietal craniotomy and resection of the single brain metastasis with ultrasound-guided needle localization. Pathologic examination of the specimen revealed an adenocarcinoma, consistent with esophageal cancer. The patient had an uneventful postoperative course and returned home able to ambulate but with persistence of a left upper extremity paralysis. This was followed by whole-brain irradiation. As of July 15, 1997, 33 months from the initial diagnosis and 13 months since the resection of the brain metastasis, the patient had shown no evidence of recurrence, but had developed seizure activity controlled medically and persisted with left upper extremity paralysis and mild weakness of lower extremity.

Case 2

A 59-year-old black woman presented on June 7, 1995, with dysphagia and atypical chest pain. She underwent an upper gastrointestinal (GI) swallow and EGD which revealed a squamous cell carcinoma of the distal esophagus. CT scan showed a localized tumor that was 2.8 cm in diameter, although the upper GI series showed the tumor extending along the wall of the esophagus for a distance of approximately 6 cm. There was no evidence of disease in the liver, chest, or lymph nodes. She was considered to be a clinical stage T3N0M0 (stage IIa). She received preoperative chemoradiotherapy, consisting of two courses of 5-fluorouracil at 1700 mg by continuous IV infusion daily for four days and Cisplatin at 125 mg on day one during the first and fifth week of radiation. A total radiation dose of 4500 cGy in 25 fractions over five weeks was delivered, 3240 cGy to the esophagus, mediastinum, and celiac axis and 1260 cGy to the distal esophagus. She had complete response to the chemoradiotherapy by CT scan. During the therapy she developed lower GI bleeding. A colonoscopy revealed diffuse diverticulosis and a small lesion on the transverse colon. This lesion was determined to be a stage I colon cancer. On September 5, 1995, she underwent a transhiatal esophagectomy and transverse and descending colectomy, with coloproctostomy. Intraoperative blood loss was 300 ml, with no blood products needed. No residual tumor was identified in the esophagus. The colon cancer was a T1N0M0 (stage I). The postoperative course was complicated by respiratory insufficiency that required endotracheal intubation for several days. She had persistent diarrhea that eventually improved with medical therapy. Her total length of hospital stay was 36 days. She later developed an anastomotic stricture that required several dilations. As of July 15, 1997, she continued to do well without evidence of recurrence, 25 months since the initial diagnosis. Her weight has remained stable and Karnofsky performance status is 100%.

Surgical technique for transhiatal esophagectomy

Transhiatal esophagectomy includes an abdominal and neck incision with resection of the esophagus without thoracotomy. The left side of the neck is the site of access to the proximal esophagus.

Abdominal access is provided by an upper midline incision. The abdomen is explored for metastatic disease. Mobilization of the stomach begins along the greater curvature, staying outside of and carefully preserving the right gastroepiploic arcade, which is the major blood supply for the gastric interposition. A pyloromyotomy or pyloroplasty is performed.

The proximal hepatic lymph nodes and left gastric lymph nodes are mobilized and the left gastric artery is taken at its origin from the celiac axis. The peritoneum overlying the hiatus is opened, sweeping all nodal and soft tissue down onto the specimen. Mobilization of the stomach is now complete.

With the use of a mechanical retractor to elevate the lower end of the sternum and the costal margins, and deep manual retractors to open the diaphragmatic hiatus, excellent exposure to the posterior mediastinum is provided. Under direct vision, a dissection including the pericardium anteriorly, the right and left mediastinal pleura laterally, and all posterior tissue down to the aorta can be developed around a bulky lower esophageal tumor. Once the dissection has extended for several centimeters beyond the upper margin of the tumor, the dissection is shifted to a plane just outside the esophageal wall, mobilizing the esophagus from the periesophageal soft tissues. Usually, the avascular plane anterior to the esophagus is bluntly developed as a first step, and then a similar plane is developed by blunt finger dissection posteriorly. Then, under direct vision, the lateral "stalks" are first clipped and then divided (**Figures 1, 2**). In this manner, dissection proceeds from the hiatus to the level of the carina. Once the transhiatal dissection has reached the level of the carina, dissection is interrupted until the cervical mobilization has advanced well into the superior mediastinum. An incision is made parallel to the anterior border of the left sternocleidomastoid muscle. The esophagus is encircled low in the neck, identifying the left recurrent laryngeal nerve and gently keeping it in contact with the trachea as it is mobilized away from the esophagus.

Blunt finger dissection then proceeds down the esophagus into the superior mediastinum. Injury to the left recurrent laryngeal nerve, the membranous portion of the trachea, the azygous vein, and the thoracic duct is avoided by maintaining the dissection in direct contact with the esophageal wall, brushing the surrounding soft tissue away from the esophagus. Simulta-

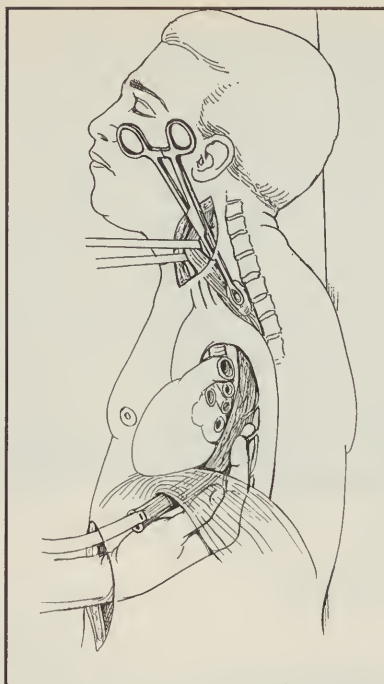


Figure 1. Transhiatal esophagectomy: Using blunt dissection the esophagus is mobilized, preserving the vascular pedicles intact.

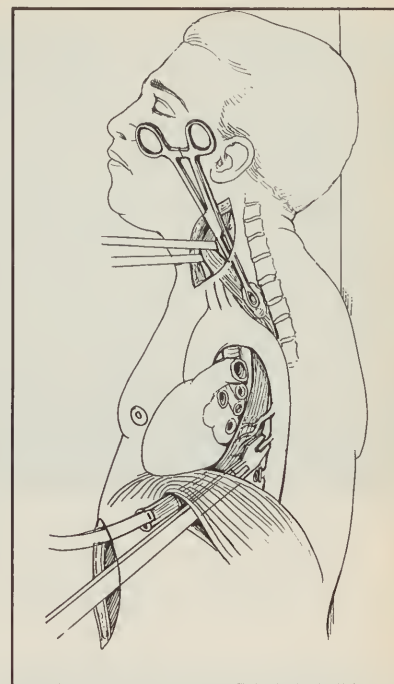


Figure 2. Transhiatal esophagectomy: Using the abdominal and cervical approach without thoracotomy, the vascular pedicles are clipped and divided under direct vision.

neously, the transhiatal dissection is carried upward and the cervical dissection downward until the upper and lower dissections meet (**Figure 1, 2**).

Arterial hypotension is frequently encountered during the blunt mediastinal dissection, and the patient's blood pressure is continuously monitored. During the typical transhiatal mediastinal dissection, blood loss is limited to 500 cc or less; rapid and persisting bleeding may indicate a tear of the azygous vein and requires conversion to transthoracic exposure for delineation and control of the bleeding site. We have not encountered this complication in our personal experience; the majority of the dissection is accomplished by direct vision and clipping of vessels, thus limiting the intraoperative blood loss.

After the esophagus has been freed completely from its mediastinal attachments, it is divided in the neck with the TA-30 stapler. Provided that an adequate proximal margin is ensured, several centimeters of the upper thoracic esophagus should be preserved since this will facilitate the subsequent anastomosis and may also enhance postoperative swallowing. The thoracic esophagus is then extracted from the mediastinum and delivered into the abdominal cavity and onto the anterior chest wall. Using five to eight applications of the GIA stapler, a gastric tube is created based on the greater curvature of the stomach and the right gastroepiploic vessels, thereby excising the cardia, lesser curvature of the stomach, and the attached left gastric, hepatic, and celiac lymph nodes (**Figures 3, 4, 5**).



Figure 3. Using five to eight applications of the GIA stapler, a gastric tube is created based on the greater curvature of the stomach. A pyloroplasty is also performed.

The gastric tube is delivered to the neck, taking care to avoid torsion. Anastomosis is done in a single layer of absorbable suture (**Figure 6**). Abdominal and cervical wound closure is performed in the routine fashion, and chest tubes are placed if required by entry into either the right or left pleura.

Discussion

The prognosis of esophageal cancer is poor, with an overall five-year survival rate of 25% following surgical resection and 17% following radiation therapy as a single modality therapy.⁶ Radiation alone provides good palliation for more than 80% of patients, but up to 50% will develop a recurrence of dysphagia.

Restoration of swallowing is critical for providing good quality of life to patients. Due to the rare recurrence of dysphagia after surgery, for surgically resectable patients, surgery alone has been considered the standard of care. The extent of surgical resection has not been shown to influence survival. In the series of Skinner et al, with a radical resection through an open thoracotomy the five-year survival rate was similar to that of Orringer et al, who used a transhiatal approach. The five-year survival rates were 18% and 17%, respectively.^{7,8}

Traditionally, Japanese surgeons have advocated more extensive resections to include more extensive lymphadenectomies (i.e., the cervical, thoracic, and celiac regions). Kato et al., reported 30% five-year survival for patients in whom the cervical lymph nodes were the only site of cancer spread.⁹ Using a subtotal esophagectomy and three-field lymph node dissection, Baba et al., reported a 30% five-year survival of 30% in patients with stage III disease.¹⁰ Recently, Altorki and Skinner reported cervical lymph node metastases to be present in 20% to 30% of patients undergoing three-field lymphadenectomy.¹¹ No prospective randomized trials have demonstrated the benefit of this aggressive approach. Few surgeons have been able to duplicate

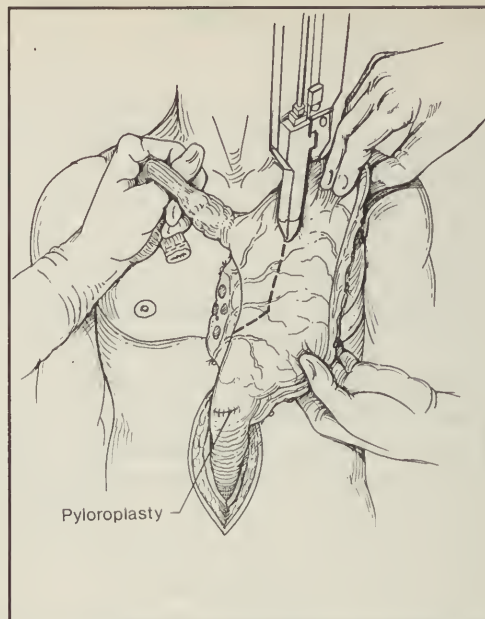


Figure 4. Case 2 — the gastric tube is being created using the GIA stapler.

the reported survival statistics of patients undergoing esophagectomy for carcinoma in Japan. Many of us feel that extrathoracic nodal spread is an indicator of incurability, and that other modalities are needed to decrease the incidence of distant disease.¹²

One major trend in the surgical approach to esophageal resection over the past two decades has been the switch from a transthoracic esophagectomy (TTE) to a transhiatal esophagectomy (THE). The THE offers similar disease-related survival and lower postoperative morbidity and mortality rates, especially for elderly patients and high operative risk patients (ASA risk 3 or 4).¹³ Most of these patients with esophageal cancer are considered high operative risks. The complications of THE are mainly cardiopulmonary, with an incidence of 13% to 81% of respiratory complications and 3% to 44% of cardiac complications. Mortality is 2.5% to 16% in a survey of several large series.¹⁴ Strictures at the anastomosis are more common in the THE approach. The retrospective study of Bolton et al., demonstrated a 0% mortality for THE compared with a 17% mortality for thoracotomy. In this study the two-year survival rate was 40% for THE and 18% for TTE.¹³ In the senior author's experience with THE, the mortality rate has remained 0% as previously reported, regardless of the preoperative ASA status.¹³ But the cardiopulmonary morbidity is high as illustrated in Case 2.

The role of chemotherapy in esophageal cancer remains investigational and, as a single treatment modality, has shown no impact on survival. Chemotherapy has been used in three situations as palliative treatment of advanced esophageal cancer, where it has at best limited benefit. Chemotherapy has been used in conjunction with irradiation and without surgery. In this setting, the combined



Figure 5. The gastric tube has been constructed and is ready to be delivered to the neck.

therapy has been shown to be better than radiation alone. However, the recurrence or persistent locoregional disease was substantial. The 24-month survival was 10% for the radiation group and 38% for the combined modality group. However, for the combined therapy, the median survival was only 12.5 months. In addition, 44% of the initial recurrences of the combined therapy group were within the radiation field.¹⁵

Given the poor prognosis of all these therapies a new therapeutic regimen is needed. One that shows promise is a combined modality approach, that is, neoadjuvant chemoradiotherapy prior to esophagectomy. The objective of this approach is to reduce the size of the tumor, improving resectability rates, and to decrease the incidence of microscopic metastatic disease. Multiple chemotherapeutic regimens have been tried. Cisplatin-based combinations appear to have the best responses. An overall response of 40% to 80% and a pathological complete response of 24% have been seen when combined chemotherapy and radiotherapy are added in the neoadjuvant setting. Patients who had a pathologic complete response have demonstrated increased median survival of 11 to 29 months.¹⁶

Vogel et al., retrospectively analyzed 125 patients with either adenocarcinoma or squamous cell carcinoma who underwent either surgery only or preoperative chemotherapy and/or radiation therapy followed by surgery. Ninety-eight patients underwent potentially curative resections (transhiatal esophagectomy in 70 and transthoracic esophagectomy in 28). There were no differences in overall mortality or surgical complications in either group. Thirty-two percent of patients in the combined modality group had a complete pathological response. In this group with complete response the five-year survival was 57%. Thirty-four percent of patients were down-staged to microscopic residual tumor, with a three-year survival rate of 31%. Overall, five-year survival in the adjuvant therapy plus surgery group versus the surgery-only group was 36% and 11%, respectively ($p=0.04$). In

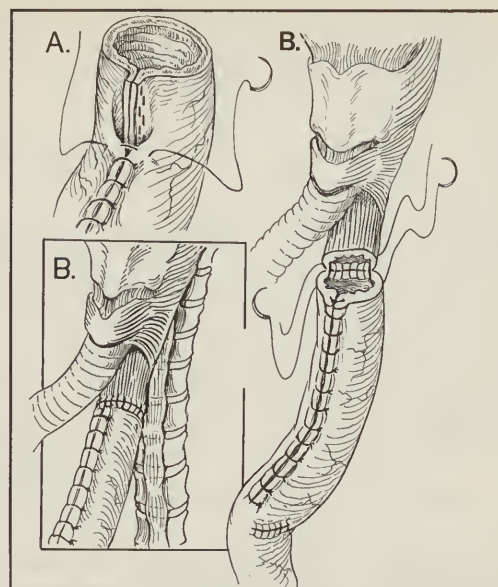


Figure 6. A: The GIA stapler line is oversewn with 3-0 silk suture.

B: An esophagogastric anastomosis is performed in the neck using a single layer of absorbable suture.

the unresectable patients, chemoradiotherapy did not have a positive effect on survival.¹⁷

A series from the University of Michigan, using preoperative chemoradiation followed by transhiatal esophagectomy, demonstrated a 24% complete pathological response rate. Complete-response patients had a median survival of 70 months and 60% were alive at five years, while the patients who had residual tumor in the resected esophagus had a median survival of 26 months with 32% alive at five years. The survival of the group treated with neoadjuvant chemoradiotherapy compared with previous studies with transhiatal esophagectomy suggests that neoadjuvant therapy improves survival.¹⁸

Historically, squamous cell carcinoma has been considered more responsive to radiotherapy and chemoradiotherapy. But studies have shown that both histological types, adenocarcinoma and squamous cell carcinoma, have similar response rates. In the study of the University of Michigan, the five-year survival rates were 34% and 31%, for patients with adenocarcinoma and squamous cell carcinoma respectively.¹⁸

The role of surgery as part of chemoradiation protocols has been questioned by some investigators. The Michigan data reveal that surgery is an important component of this approach. Following chemoradiotherapy, approximately one-third of the patients had persistent tumor in the resected specimens, but following resection these patients survived five years and no local recurrences were subsequently seen.¹⁸ Esophageal resection not only provides the ultimate means for assessing primary tumor response but also offers the best means of providing local disease control and potential prolongation of life.¹⁹

In the two cases presented, one had a partial response and the other had a complete response. Both patients had excellent local

control of their disease. The patient with adenocarcinoma had a brain metastasis. Solitary metastatic disease to the brain without other sites of recurrence should be treated with surgery followed by irradiation. This approach gives the best results. As seen in this patient, he remained free of disease for 13 months after the resection of the brain metastasis.

The toxicity of simultaneous chemotherapy and radiation therapy is certainly more substantial and frequent than that seen with either modality alone. Although myelosuppression, including leukopenia and thrombocytopenia, with resulting episodes of febrile neutropenia and infection, occurs rarely, significant gastrointestinal side effects, including nausea and vomiting, oral stomatitis, and esophagitis, are frequent problems. Nutritional support is of vital importance to assure that the patient tolerates and completes the therapy. Other more severe but infrequent esophageal problems include perforation, fistula formation, and fibrosis and stricture. In addition, depending on the specifics of radiation port and chemotherapeutic drugs, pulmonary and skin toxicity might be enhanced.⁶ The chemotherapy related mortality has been from 3% to 6% and surgery from 0% to 16%. In experienced hands this mortality can be kept under 5%. This multimodality approach needs to be performed by experienced physicians. Radiation, medical, and surgical oncologists work together to assure that the morbidity and mortality are kept at the lowest possible levels. An experienced support team of nurses, nutritionists, and respiratory and critical care personnel are also needed to assure good outcome.

The theoretical advantages of such neoadjuvant therapy include an increased rate of resectability of locally extensive tumors, improved survival, improved control of micro-metastasis, and the ability to administer higher drug dosages than are normally tolerated in the postoperative setting.²⁰ In addition, it has been found that preoperative chemoradiation results in significant down staging, increased survival, and the possibility of local and regional lymph node sterilization.

Esophagectomy in this setting can be accomplished with acceptable morbidity and mortality. There has not been an increase in postoperative morbidity or mortality after pretreatment with chemoradiotherapy. Unfortunately, to date, no randomized trial has documented a survival benefit of preoperative therapy compared with operations alone. Prospective randomized control trials are underway and will guide future management of esophageal cancer. New technological advances with transesophageal ultrasonography will help us to better stratify these patients in future clinical trials.

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ONCOLOGY . . . TODAY

Diet and cancer

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ABSTRACT

Unhealthy diets are a major cause of cancer. Extensive scientific research implicates three components of diet as being particularly important in the etiology of various cancers: dietary fat, fruits and vegetables, and fiber. The evidence linking these components to the incidence of numerous cancers warrants physicians making strong dietary recommendations to reduce their patients' risk of developing cancer.

Introduction

Cancer is responsible for approximately 23% of all deaths in the United States.¹ Although it is not widely recognized, it is estimated that 35% or more of all cancer deaths are attributable to unhealthy dietary practices.² This represents more cancer deaths than are attributable to any other cause.²

Elucidating exactly which components of diet are most important in the etiology of cancer has been a difficult, time-consuming process. Dietary studies are complex and fraught with numerous pitfalls.³ People consume many different foods and different amounts of each of these foods. Capturing the variety, as well as the quantity, of foods habitually eaten is not an easy task. Furthermore, people's diets often change over time. No dietary assessment instrument thus far developed

gives a complete and accurate picture of a person's diet.

Almost every study of diet and cancer has used different dietary assessment techniques, different lengths of time for which dietary information was collected, different lists of foods whose consumption was assessed, different means of assessing the portion sizes, different time periods prior to the development of cancer during which diet was assessed, and different ways of reporting results. Therefore, it should not be surprising that some studies have produced conflicting results regarding relationships between the dietary factor being studied and the risk of developing a particular type of cancer.

Despite all of these difficulties, a large, rather consistent body of evidence now exists linking certain dietary components in a dose-response relationship with the risk of developing cancer. Dietary components that are associated with a higher risk of developing cancer include fat and smoked, salted, and pickled foods. Those that are associated

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with a lower risk of developing cancer include fruits, vegetables, and fiber.⁴

Table 1 summarizes the most important dietary factors from the perspective of population attributable risk that may play a causal role in the etiology of common cancers in the United States: fat, fruits and vegetables, and fiber. The evidence of this role consists of animal, international correlation, migrant, case-control, and cohort studies. This article briefly reviews some of the evidence linking these important dietary components and cancer. It presents mechanisms through which these components might act. It concludes by presenting dietary recommendations for physicians to provide to patients.

Fat

A large body of data available from experimental animal studies implicates dietary fat in carcinogenesis.^{5,6} High levels of fat are associated with increased numbers and incidence of tumors at several sites, including the mammary glands and the colon. Consumption of either polyunsaturated fats or saturated fats is associated with increased incidence of tumors. The tumors occur at the highest rates when dietary fats are given following exposure to a known carcinogen, suggesting that the role of fats is primarily that of a cancer promoter.

In human studies, dietary fat has been associated with many forms of cancer. One of the most compelling pieces of evidence comes from international correlation studies. In one such study, food disappearance data available from the Food and Agricultural Organization for 40 different countries from 1964 to 1966 were correlated with age-adjusted mortality rates for various can-

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cer sites during the same time period.⁷ Very high correlation coefficients (CC) ranging from .89 to .94 were found for cancers of the breast, colon, and prostate. The coefficients were also quite high, ranging from .50 to .78 for leukemia and cancers of the ovary, lung, pancreas, skin, and bladder. Age-adjusted mortality rates for some cancers were more than six times higher in countries with high fat consumption (e.g., the United States) than in countries with low fat consumption (e.g., Japan).⁷ Other correlation studies have found similar results.^{8,9}

Case-control and cohort studies have shown varied results. Most showed higher incidence and mortality rates of various cancers, particularly cancers of the breast, colon, and prostate in individuals consuming higher amounts of fat or surrogates such as red meat.¹⁰⁻¹⁸ Others did not find differences in these rates;¹⁹⁻²¹ failure to detect such differences may have been due to studying populations with too similar a range of dietary fat. Most migrant studies have shown higher rates of cancer in populations who move to countries consuming large amounts of fat and adopt this practice compared with populations who remain in their native countries and consume less fat.^{22,23}

Fruits and vegetables

More than 200 case-control and cohort studies have shown an association between higher levels of fruit and vegetable intake and reduced risk of developing a large variety of cancers. Cancers for which there is a reduced risk associated with fruit and vegetable consumption include the

pharynx, esophagus, oral cavity, stomach, pancreas, colon, rectum, prostate, larynx, lung, bladder, endometrium, cervix, and ovary.²⁴⁻³² The studies indicate that a variety of fruits and vegetables are associated with a reduced risk of developing cancer, including dark green, yellow, and orange fruits and vegetables, cruciferous vegetables, dried fruits, berries, beans, tomatoes, and carrots.

The evidence is particularly strong for epithelial cancers, especially those of the digestive and respiratory tract. A recent review of 156 studies of dietary influences on such cancers revealed that 128 had shown a statistically significant protective effect of fruit and vegetable consumption.²⁴ These studies were conducted in 17 countries with very diverse populations, including the United States, Netherlands, India, and China. Persons who consumed fewer fruits and vegetables generally had a risk of developing cancer at least twice as high as those who consumed more.

Fiber

A somewhat smaller number of studies have been conducted focusing on the relationship between dietary intake of fiber and risk of cancer. The major link which has been found is between consumption of fiber and risk of colon cancer.

Greenwald reviewed all of the major studies that assessed this relationship.³³ Of the 40 studies reported in his article, 32 showed a negative association between the amount of fiber consumed and either incidence or mortality from colon cancer; that is, the more fiber consumed, the lower the risk. This review clearly demonstrated that the preponderance of evidence linked fiber consumption to colon cancer. Subsequent studies have supported this link.

Discussion

Most studies have found strong, consistent relationships between the amount of dietary fat, fruits, vegetables,

Table 1. Strength of the evidence linking dietary factors and various cancers

Cancer	Fat	Fruits & Vegetables	Fiber
Lung	◆	■	
Colorectal	◆	■	■
Breast	◆		
Prostate	◆	■	
Pancreas	◆	■	
Oral Cavity		■	
Pharynx		■	
Esophagus		■	
Larynx		■	
Stomach		■	
Ovary	◆	■	
Endometrium		■	
Cervix		■	
Bladder	◆	■	
Leukemia	◆		

◆ Strong evidence linking higher consumption levels with an increased risk of developing this cancer.

◆ Suggestive evidence linking higher consumption levels with an increased risk of developing this cancer.

■ Strong evidence linking higher consumption levels with a reduced risk of developing this cancer.

■ Suggestive evidence linking higher consumption levels with a reduced risk of developing this cancer.

and fiber consumed and the risk of developing and/or dying from these cancers. The totality of evidence from these studies meets the other classic criteria for determining that these dietary components are causally linked to cancer: temporal relationship of the exposure to the outcome of interest, independence of the association, and coherence of the data among different species and study designs.

A central question of biologic plausibility remains: Are there logical mechanisms through which these dietary factors might operate? Animal studies indicate that dietary fat results in depressed immunologic function.³⁴ Dietary fat also increases estrogen in women and testosterone levels in men.^{35,36}

Another means by which dietary fat may be associated with an increased risk of cancer relates to the manner of cooking. A significant increase in fecal mutagens has been found in those consuming either fried or charred meat or fish.³⁷ Mutagens similar to those released in fried or charred meat products have been shown to cause cancer in animals.

The situation with regard to colon cancer is slightly more complex. Higher fat diets are associated with changes in the fecal flora, increases in the activity of certain bacterial enzymes such as β -glucuronidase, and increases in fecal bile acids.³⁶⁻⁴¹ β -glucuronidase may increase the level of active carcinogens in the colon by breaking down various compounds excreted in the bile as glucuronide conjugates. Fecal bile acids have been shown to be tumor promoters in animal experiments; they have also been found in higher concentrations in colon cancer patients as compared with controls. Bacterial breakdown of some bile acids can also result in the production of secondary bile acids that are tumor promoters.

There also are plausible biochemical mechanisms for the protective effects of fruits and vegetables. Fruits and vegetables contain vitamin A, vitamin C, vitamin E, folate, carotenoids and other antioxidants, fiber, and non-nutritive substances such as dithiolthiones, flavonoids, glucosinolates, indoles, isothiocyanates, phenols, d-limonene, and allium compounds.⁴² Each of these substances may play a role in reducing the risk of developing cancer. More likely, it is a combination of these and other unknown factors that confer protection.

Dietary fiber probably reduces risk through at least two mechanisms. First, increased dietary fiber is associated with decreased transit time through the colon, thereby reducing the length of time the colon is exposed to any carcinogens. Second, increases in dietary fiber result in greater stool bulk, thereby decreasing the concentration of bile acids and any other carcinogens moving through the colon.

Conclusions

A large body of evidence points to the important role of certain dietary habits and the risk of developing cancer. This evidence suggests that the level of dietary fat is a major determinant of the risk of developing cancers of the breast, colon, and prostate, with suggestive evidence for several other types. Low consumption of fruits and vegetables is strongly associated with increased risk of developing at least eight types of cancer, with suggestive evidence for another six types of cancers. Low levels of dietary fiber are associated with increased risk of developing colon cancer.

Over the past 20 years, many scientific organizations in the United States have reviewed the available evidence and have issued dietary recommendations for the prevention of cancer.

These organizations include the National Cancer Institute, the Department of Health and Human Services (U.S. Surgeon General's Office), the National Academy of Sciences, the American Cancer Society, and the American Health Foundation. Their recommendations to the public have been remarkably similar: reduce consumption of dietary fat to $\leq 30\%$ of calories, increase consumption of fruits and vegetables to five to nine servings daily, and increase dietary fiber to 20 grams to 30 grams per day.

Physicians should make similar recommendations to all patients ages two and above. Despite modest improvements in dietary patterns in the United States over the past 30 years, most Americans are still not meeting these dietary guidelines. The most recent data from NHANES III, a national sample of dietary practices, found that more than 75% of whites and blacks in the United States were consuming a diet high in fat, low in fruits and vegetables, and low in fiber.⁴³

Most patients would benefit from altering their diet to one lower in fat and higher in these other substances. They will thereby decrease their risk of developing certain cancers with the added benefit of decreasing the risk of developing coronary heart disease.⁴⁴ Physicians can provide great assistance to their patients, both by motivating them to make such changes and by giving them specific suggestions for what kinds of foods and food preparation techniques they should be adopting.

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Psychotropic medication use in people with developmental disabilities

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Abstract

Psychotropic medications are frequently used to treat undesirable behaviors in persons with developmental disabilities. Successful use of these drugs is dependent on accurate assessment of the psychiatric disorder or behavioral problem. Treatment of aggression and self-injurious behavior and the use of antipsychotics, antidepressants, mood stabilizers, anxiolytics, beta-blocking agents, and naltrexone will be discussed.

Introduction

Psychotropic medications are frequently employed in persons with developmental disabilities to treat psychiatric disorders such as schizophrenia, depression, bipolar disorder, and anxiety disorders much as they are used in the general population. The prevalence of psychotropic medication use in this population varies considerably depending on the setting. The mean prevalence rate reported by Baumeister et al., is 57%, 41%, and 22% in institutions, community, and school settings, respectively.¹ Antipsychotic drugs are the most frequently used.^{1,2}

Psychotropic drugs are also often employed in persons with developmental disabilities to control or manage "maladaptive" behaviors including aggression, hyperactivity, self-injury, and disruptive or inappropriate social behavior.^{1,2} These are vague and frequently poorly defined behaviors rather than psychiatric syndromes. The use of drugs for these behaviors is controversial. The efficacy of psychotropic medications for these purposes is poorly documented and ill defined.

The key to appropriate use of psychotropic medications in people with developmental disabilities is accurate assessment of the psychiatric disorder or behavior problem. It is crucial to rule out environmental causes of the problematic behavior prior to initiation of pharmacologic therapy.³ When other causes cannot be found, or it is difficult or impossible to give a psychiatric diagnosis to a given individual with a developmental disability, target symptoms should be specifically defined. Length of a medication trial depends on the type of drug used and its purpose. Most antipsychotic medications take at least a week to reach steady

This article is the fourth in a series that will explore various aspects of treating patients with developmental disabilities.

state and may take longer to reach maximum therapeutic effect. Other drugs with much longer half-lives, such as fluoxetine, may take up to a month to reach steady state and will require a longer trial. Medication should be maintained at least two to three months at therapeutic doses or levels to adequately determine efficacy. A drug should be discontinued if there is no evidence of improvement or if side effects outweigh its benefit.

Aggression and self-injurious behavior

People with developmental disabilities are frequently referred to psychiatrists for treatment of aggression and self-injurious behavior (SIB).⁴ SIB is defined as any chronic, repetitive behavior that results in physical injury to a person's own body and is more common in those with severe and profound developmental delay. Aggression may take many forms, including physical assault, verbal aggression, or property destruction. Self-injurious or aggressive behavior may be caused by inability to communicate and/or environmental influences; therefore, examination of environmental factors is crucial. The cause of problem behavior may also be a result of a medical illness, and a thorough review of potential medical causes, including a physical examination, should be performed.³ Aggression and SIB may be caused by pain from dental problems, dysmenorrhea, gastrointestinal upset, or otitis media. Aggression and self-injury may also result from psychiatric disorders such as mood or psychotic disorders.

Antipsychotic medications are frequently employed to treat these behaviors. There are reports in the literature that these drugs will decrease SIB and aggression in people with developmental disabilities. However, it is unclear that these effects are specific.⁴ The sedative effects of antipsychotic medication may simply suppress all behavior. Additionally, these agents may decrease learning in some developmentally delayed individuals.⁴

Side effects of the antipsychotic medications must also be considered when they are used in this population. People with developmental disabilities experience the same problems as others in the general population. Identification of side effects may be more difficult due to communication problems. Akathisia, a subjective sense of restlessness and an inability to sit still, is frequently unrecognized and may be confused with an increase in maladaptive behaviors or hyperactivity.⁵ Tardive dyskinesia, akathisia, and dystonia are seen in people with developmental disabili-

ties as a result of antipsychotic administration. Symptoms of the tardive syndromes may mimic or be confused with stereotypy or an underlying neurologic problem. A baseline assessment and follow up are crucial.

The atypical antipsychotic medications clozapine, risperidone, and olanzapine may be of use in selected individuals that exhibit aggressive behavior. Clozapine has been reported to improve aggressive and self-injurious behavior in people with developmental disabilities.^{6,7,8,9} Risperidone has also been reported to reduce aggression and self-injurious behavior.^{10,11} No published reports on the effectiveness of olanzapine or sertindole (due to be released) could be found.

There are numerous reports on the use of naltrexone to treat self-injurious behavior in developmentally delayed people. Theoretically, a person who engages in self-injury releases endogenous opioids and receives positive reinforcement from the narcotic effect of these endogenous substrates.¹²⁻¹⁴ Naltrexone blocks the effect of exogenous narcotics and endogenous opioids. Most of the literature describing the use of the opioid antagonists comes from case reports and open trials. It appears that some individuals may benefit from the use of naltrexone, but not everyone who engages in self-injury will benefit from the drug. A trial of naltrexone may be warranted in an individual for whom no apparent environmental, physical, or psychiatric cause is identified. The dose of naltrexone used for this purpose is usually 1.0 mg/kg/day to 2.0 mg/kg/day.¹²⁻¹⁵

Beta-blocking medications such as propranolol, metoprolol, and nadolol have been reported to decrease aggression and self-injury in people with developmental disabilities.¹⁶⁻¹⁹ However, most of the published reports are also case reports and open studies. Some persons may benefit from the use of these drugs, especially if the aggression and SIB are caused by neuroleptic-induced akathisia.¹⁵ Doses of these agents reported to treat these behaviors in developmentally delayed individuals vary from 200mg to 1000 mg per day.¹⁹

Many other drugs such as antidepressants, anxiolytics, and anticonvulsants have been employed to treat or control aggressive and self-injurious behavior. Unfortunately, none of these drugs specifically treats these behaviors. They may be effective in a specific individual; however, there is no one medication that will eliminate these maladaptive behaviors.

Psychotic disorders

Persons with developmental disabilities suffer from the same psychotic disorders as the general population. Manifestation of the illness may be different in this population. The person's ability to communicate and verbal and cognitive sophistication must be evaluated prior to establishing a diagnosis.

The choice of a medication should be based on the individual's health status and medication history. A previous response to a medication may predict a response. Medications such as thioridazine (Mellaril), chlorpromazine (Thorazine), loxapine (Loxitane), and clozapine (Clozapine) may lower the seizure threshold in individuals with a seizure disorder and should be used with caution. The presence of extrapyramidal symptoms such as pseudoparkinsonism and akathisia may make the higher potency medications (haloperidol, thiothixene, fluphenazine) less desirable. Lower potency medications (chlorpromazine, thioridazine, mesoridazine) cause more sedation, constipation, urinary retention, and cognitive impairment.

Antipsychotic medications should be used at the lowest dose possible. This is important not just for side effect and therapeutic reasons, but also satisfies state and federal guidelines for using the least restrictive intervention.²⁰

Some literature suggests that the newer medications may be beneficial in this population.⁶⁻¹¹ Risperidone, used at low doses, may have fewer side effects than the traditional agents and is well tolerated in this population.^{10,11} Clozapine should be reserved as a last line agent because of its potential toxicity. Currently, there are no published reports of olanzapine use in this population; therefore, its place in therapy is not yet established.

Mood disorders

Mood disorders may also present differently in people with developmental disabilities than in the general population. Again, depending on the functioning level of the individual, it may be difficult to make definitive diagnosis of bipolar disorder or depression. A history of behavioral cycling, irritability, and sleep problems may indicate the presence of a mood disorder.²¹ Changes in adaptive functioning, self-injury, and aggression may also be present in people with mood disorders.²²

Valproic acid, carbamazepine, and lithium are effective medications for treating people with bipolar disorder regardless of level of functioning. However, the literature is sparse regarding the use of these drugs in treating people with developmental disabilities. One open study and several case reports show good results using valproic acid in people with developmental disabilities for treatment of what appears to be affective disorders.²¹⁻²⁵ Serum valproic acid levels ranged from 64 µg/ml to 144 µg/ml in these reports. Valproic acid is usually well tolerated, however, many people experience gastrointestinal upset and sedation. Thrombocytopenia is associated with the use of valproic acid and should be periodically monitored. Other side effects that have been reported in people with developmental disabilities include pancreatitis and cholecystitis.²⁶

The Food and Drug Administration has not approved carbamazepine for use in bipolar disorder; however, it is effective in treating people with this disorder.²⁷ Carbamazepine has a reputation as having "antiaggressive" properties, however, there is little literature to support this concept. Carbamazepine appears to be most effective in people who display symptoms consistent with bipolar disorder and also have a seizure disorder.²⁸⁻³⁰ Carbamazepine has many side effects, including gastrointestinal upset, ataxia, and diplopia.³¹ These side effects can be minimized by starting the drug at low doses and increasing slowly to the desired serum concentration. Other side effects that need to be monitored include leukopenia, jaundice, and electrolyte imbalances. Because of the potential for agranulocytosis and leukopenia, concomitant administration of carbamazepine and other drugs that can also cause bone marrow suppression should be avoided.³¹ Clinically significant drug interactions occur with carbamazepine. This drug is a potent inducer of liver enzymes in the cytochrome P-450 system and reduces the serum concentrations of other drugs metabolized in that system. Drugs that are significantly affected by carbamazepine include, but are not limited to, phenytoin, phenobarbital, primidone, valproic acid, antipsychotic medications, oral contraceptives, and theophylline.³¹

Lithium has been extensively used in persons with developmental disabilities for many purposes.³²⁻³⁴ Again, as with the other drugs used to treat people with mood disorders, lithium appears to be most effective in people who have cyclical behavioral symptoms. Pary reviewed the literature up to 1991 for the use of lithium in this population and provided the following guidelines: Serum concentrations of at least 0.5 mEq/ml to 1 mEq/ml should be obtained unless the person responds or side effects prevent achievement of that level. The trial should last for at least six to eight weeks at adequate serum concentrations before concluding that the person will not respond.³² Lithium can cause many side effects.³⁵ People taking this drug frequently have gastrointestinal upset, tremor, muscle weakness, and fatigue. Hypothyroidism, leukocytosis, polyuria, and polydipsia can also occur. Lithium is excreted exclusively through the kidney and this must be taken into consideration in people with impaired renal function.

Antidepressant medications have also been used to treat people with developmental disabilities. The amount of formal research with these drugs in this population is limited. Tricyclic antidepressants have been reported to be beneficial in persons exhibiting symptoms of depression, psychosis, and tantrum behavior, and in those who have a high social age.³⁶ The tricyclic antidepressants have significant side effects, including dry mouth, urinary retention, constipation, and blurry vision. They also cause significant sedation, hypoten-

sion, cardiac arrhythmias, and a decrease in the seizure threshold. The tricyclic antidepressants may also cause deterioration in symptoms of irritability, lethargy, social withdrawal, and hyperactivity.³⁷ These side effects may limit their use in people with developmental disabilities.

The selective serotonin reuptake inhibitors (SSRIs) are a relatively new group of antidepressants. This class of drugs includes fluoxetine, paroxetine, sertraline, clomipramine, and fluvoxamine. These drugs have been used to treat people with developmental disabilities who appear to have symptoms of depression.³⁸⁻⁴² Symptoms of depression may present in a person with developmental disabilities as social withdrawal, sleep and eating changes, self-injury, and aggression.³⁸ The SSRIs are not without side effects. Two studies report increased irritability, hyperactivity, agitation, anorexia, and insomnia in developmentally delayed people who received fluoxetine.^{41,42} Other side effects include gastrointestinal upset and sexual dysfunction. These drugs may be safer and less toxic than the tricyclic antidepressants. The clinician must be aware, however, of the potential for worsening of behavior problems, especially with fluoxetine. Drug interactions include significant inhibition of specific isoenzymes of the cytochrome P-450 system. Concomitant use of other serotonergic drugs may result in the serotonin syndrome. These symptoms include mental status changes, hyperreflexia, tremor, restlessness, and diaphoresis.

Antidepressants and mood-stabilizing agents are effective in treating affective symptoms in people with developmental disabilities. The choice of medication must be based on the person's presenting symptoms and medical status. Valproic acid and carbamazepine may be more effective than lithium in treating cyclical disorders in people with seizure disorders and brain damage. Tricyclic antidepressants should be avoided in people with active seizure disorders. The SSRIs may be useful in treating people with symptoms of depression and cause less toxicity than the tricyclic antidepressants, but may also exacerbate some behavioral problems.

Anxiety disorders

Anxiety disorders, including obsessive-compulsive disorder (OCD), occur in people with developmental disabilities. OCD has been described and treated in people with developmental disabilities.⁴³⁻⁴⁶ Obsessive symptoms may be difficult to ascertain due to communication limitations. However, the compulsive symptoms may be evident in ritualistic behavior. Currently, several SSRIs are indicated for use in OCD. These include clomipramine, fluoxetine, fluvoxamine, and paroxetine. Any of these may be used to treat a person who appears to

be engaging in ritualistic behavior that is interfering with activities of daily living. The same cautions and potential side effects listed above apply for the use of these drugs for this indication. Clomipramine is a tricyclic antidepressant and has the same side effects as that class of drugs. It is also of concern because of its potential to cause seizures.

Benzodiazepines have been used for "as needed" treatment for any type of behavioral disorder. The use of these drugs is declining for a few reasons: there is little evidence that they control or reduce problem behaviors in people with developmental disabilities, they may actually exacerbate agitation and aggression, and long-term use may lead to tolerance and withdrawal symptoms upon discontinuation.⁴⁷ Chemical restraint for the control of an acutely violent or agitated person is sometimes required. Benzodiazepines are useful in these situations, but should be avoided for long-term use.

Bupropion may be useful in people with developmental disabilities and anxiety. A few studies have found it to be useful in treating anxious, aggressive, and self-injurious behavior in this population.^{48,49} The doses usually employed are between 15mg/day and 45mg/day.^{48,49} Bupropion has a favorable side effect profile, however, clinicians must keep in mind that no well-controlled methodologically sound studies have assessed the effects of this drug in people with developmental disabilities.

Conclusion

Appropriate use of psychotropic medications depends on accurate assessment of the underlying psychiatric disorder. The environment can also influence behavior and must be taken into consideration when prescribing medications. Unfortunately, good controlled, methodologically sound studies are rare. Frequently, the use of psychotropic medications is empiric at best. The clinician treating these persons must be aware of the limitations of these drugs and use them only when clinically necessary and when efficacy is demonstrated by symptom resolution.

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Please fill out the following application and FAX it to Med Chi at (410) 727-5967, or mail it to **Russel Kujan, Medical and Chirurgical Faculty of Maryland, 1211 Cathedral Street, Baltimore, MD 21201.** Questions? Contact Russel Kujan, (410) 539-0872, ext. 344 or (800) 492-1056, ext. 344. Thank you.

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MARYLAND MEDICAL HISTORY

The Muse family of Maryland: To them much was given

In Roman history, “familia” was first used to denote the servants of a household, but later meant a permanent group of parents and offspring. The approach of the Medical and Chirurgical Faculty of Maryland’s 200th birthday refocuses attention on its “familia.”

Intertwined and beautifully meshed with Med Chi’s history are numerous families – and its servants. Through brotherhood, love, service, and self-sacrifice, their union has built Med Chi upon solid moral, social, and medical foundations.

Sons not infrequently follow in the footsteps of their fathers. The founding group of the faculty included 85 men of whom 17 (20%) had sons who became doctors. In the first century of its existence, Med Chi had 2073 members and 98 (4.7%) had sons who became physicians. Many of these names are familiar, including Archer, Baker, Baxley, Finney, Smith, and Trimble.¹⁻⁷ The Baxley family followed soon after the Archers and were prominent in the early days of the organization. They were supporters of the Baltimore General Dispensary and contributed 124 years of continuous service to it. Six family members, all doctors, were associated there from 1826 to 1950.

Maryland medicine has been privileged to have in its organization a family with 10 members in a direct line (Table 1). Their history begins about 1760 in Cambridge, Dorchester County, when Thomas Muse married Anne Ennals.⁸ Thomas Muse, a major in the Revolution, died in 1776. But he left two children, one of whom, Joseph (Figure 1), became a doctor and practiced in Dorchester County. Joseph was highly regarded as a physician, scientist, farmer, lecturer, and gentleman. When the Dorchester County Agricultural Society was organized in Cambridge in 1824, he was elected its first president. Joseph, the first of almost two centuries of Muse family physicians, sired three sons: Joseph E., Jr., James A., and William H.

Joseph E., Jr., the oldest son, was an expert chemist and agriculturist. In 1838, the University of Maryland recognized his extraordinary ability and conferred an honorary degree of Doctor of Medicine upon him. James A. received his medical degree from the University in 1834 and William H. earned his in 1836.

Josiah A.B. Muse (Figure 2), the son of Joseph Jr., acquired his early education in

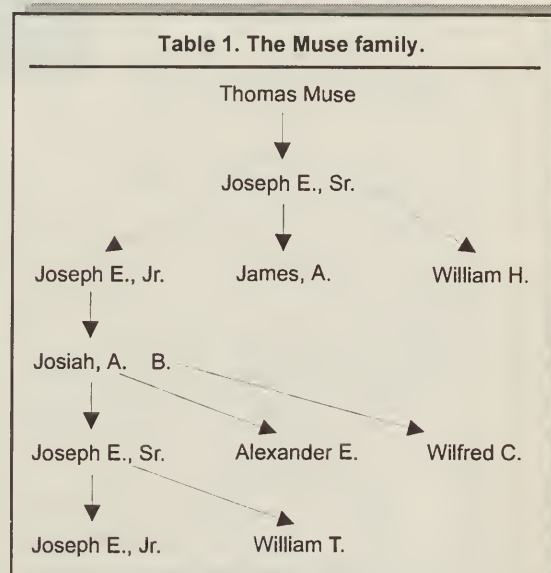


Figure 1. Joseph M. Muse, M.D.
(1776-1852)

Cambridge and Baltimore. He attended St. Timothy Hall in Catonsville and was a classmate of John Wilkes Booth. Josiah attended lectures at the University of Maryland from 1858 to 1860 and graduated on February 29, 1860 as an honor student. After the death of his father in 1856, his mother moved to Plaquemine Parish, Louisiana.

Following a short period of practice in Baltimore, Josiah was persuaded by his mother to start a practice in Louisiana. How Dr. Muse got to his destination in May 1861 is not known. He was married in New Orleans in June of that year. In a letter he describes an illness he had in July. The mystery deepens when it is recalled that Louisiana seceded from the Union in January 1861. New Orleans was retaken from the Confederacy in April 1862 after a successful rupture of the Mississippi River blockade.⁹

Josiah subsequently became a contract surgeon in the federal forces in Baton Rouge in August 1862, but shortly thereafter he was stationed in New Orleans at the United States Marine Hospital. He started at a salary of \$100 per month and later had a raise to \$200. In November 1864, he transferred to McKim General Hospital in Baltimore where the government had created a hospital in the private McKim estate in the northern part of Baltimore. After only a short stay there, he moved to Camp Parole, located a few miles from Annapolis in Anne Arundel County.¹⁰ Thousands of paroled Union prisoners were located there after 1862 until their exchange could be effected.

When the war ended, Josiah practiced in Martinsburg, West Virginia, until his death in 1876 at the age of 41. His wife then brought their three sons to Baltimore for a public school education.

Joseph E. Muse, Sr., the oldest of Josiah's sons, was born in New Orleans in 1862. He graduated from the Baltimore Medical College in 1888¹¹ and was admitted to Med Chi a year later. He was chief of clinic to the chair of diseases of women at the Maryland Medical College from 1898 to 1900 and practiced at St. Agnes and Maryland General hospitals. Subsequently, he was a member of the Baltimore City Council for six years, representing the First Branch of



Figure 2. Josiah A.B. Muse, M.D.
(1835-1876)

the 21st Ward. In the academic year of 1913 to 1914, he enrolled in the junior class at the Maryland Law School, but did not receive a degree.

Josiah's second son, Alexander E., was a graduate in pharmacy from Columbia University in 1898. He went to California for a short period and then returned to Baltimore and graduated from the Maryland Medical College in 1904. Interestingly, Alexander's brother, Joseph E., Sr., is listed as his preceptor. After his graduation from medical school, Alexander established a pharmacy on Cross Street in Baltimore. Never married, he retired in 1925 and died in 1940.

Wilfred C. Muse, Josiah's third son, graduated from the Baltimore Medical College in 1892 and practiced in Baltimore. He accidentally drowned in 1902 at the age of 32.¹²

Joseph E. Muse, Jr. (**Figure 3**), the son of Joseph E., Sr., graduated from Mt. Saint Mary's College in 1933 and then entered the University of Maryland School of Medicine, where he finished in 1937. He was a resident in medicine at St. Agnes from 1937 to 1939 and then an assistant resident at the University of Maryland from 1939 to 1940. After a brief period of private practice, he entered the Army in 1942 as a lieutenant in the Medical Corps. Assigned to an infantry regiment, he went to England and participated in the initial landing at Omaha Beach. He was wounded, evacuated to England, and eventually returned to the United States. Released from the service in 1946, he started private practice with privileges at St. Agnes, Bon Secours, University of

Maryland, and Maryland General hospitals. He was chief of the medical service at Bon Secours in 1964 and 1965.

Joseph E., Jr., and his brother, William T., lived on Hollins Street, two doors from the Mencken home. Their father, Joseph E., Sr., was the Mencken family's physician, but a strong social relationship also existed between the two families. In later years, Joseph E., Jr., provided the necessary medical care for Mencken.¹³

William T. also had his premedical education at Mt. Saint Mary's College. He received his medical degree from the University of Maryland in 1940. After an internship and three years of residency in



Figure 3. Joseph E. Muse, Jr., M.D.
(1910-1984)

surgery at St. Agnes Hospital, he became a member of the medical corps of the Army from 1945 to 1947, serving as neurosurgeon. Upon his discharge from the Army, William practiced general surgery in Baltimore, with privileges at St. Agnes, Bon Secours, and Maryland General hospitals.

Alexander Hamilton Bayly (1814 to 1892), another member of the Muse family, was born in Dorchester County. He graduated from the University of Maryland in 1835.¹⁴ He joined the faculty in 1834 and was elected vice-president in 1881 to 1882. He was the Mayor of Cambridge for over 30 years. At one time, he was also president of the Maryland State Lunacy Commission.

Samuel A. Keene, also a member of the Muse clan, was born in Dorchester County in 1843. He was educated at Mt. Saint Mary's College. A pupil of Drs. Richard McSherry and Washington C. Van Bibber, he graduated from Maryland in 1865 at the age of 22. He practiced, successively, in Dorchester County, Ellicott City, and Baltimore. He was admitted to Med Chi in 1890. He died in 1925 at the age of 82.

Bernard P. Muse, a descendant of an uncle of the original Joseph E. Muse, was born in Virginia in 1868. Bernard had a distinguished career in Baltimore medicine. A pupil of Dr. James G. Wiltshire, he graduated from the College of Physicians and Surgeons in 1888 and started a residency at the Baltimore Eye, Ear, and Throat Hospital. He then practiced in West Virginia for three years before returning to Baltimore. He was a demonstrator in surgery at the college in 1892 for one year. In 1894, he became lecturer on diseases of the eye and ear at the Baltimore University of Medicine. In the following three years, he was professor of physiology and hygiene. When the Maryland Medical College of Baltimore was created in 1898, he became dean and a member of the board of directors. He also served as professor of obstetrics for 14 years.¹⁴

Doctors in the Travers and Smith families were also related to the Muses. They graduated from a number of different eastern medical schools (Table 2).

The men of the Muse family were similar to many of their contemporaries in Maryland medicine. Some, in addition to caring for their patients, were teachers or rendered civic service. Too often, a medical group is measured only by its outstanding physicians. The rank and file who provide good care for their patients, however, are a better measure of excellence. They were and are the entrepreneurs in the movement which changed 18th and 19th century Maryland medicine to the more erudite modern medicine.

Luke, the New Testament physician, was explicit in his observation: "For unto whomsoever much is given, of him shall much be required." These men heeded the admonition

Table 2. Other members of the family who did not have the Muse name.

Name	University attended	Graduation year
John C. Travers	University of Maryland	1895
Phillip L. Travers	University of Maryland	1902
Edgar E. Travers	University of Maryland	1913
Charles F. Smith	University of Pennsylvania	1848
Fransis F. Smith	Jefferson Medical College	1854
William M. Smith, Sr.	Georgetown University	1904
William M. Smith, Jr.	University of Maryland	1953
Robert T. Fisher	University of Maryland	1977

and responded properly. They saw their duty and they did it well.

Joseph M. Miller, M.D.

Dr. Miller is a retired surgeon residing in Timonium, Maryland. ■

Acknowledgment

I am grateful to William T. Muse, M.D., for the considerable help he gave to me during the preparation of this article. He made the task much easier, and in addition, supplied the photographs.

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The Maryland Medical Journal (MMJ) is a monthly publication of the Medical and Chirurgical Faculty of Maryland. The journal's goals are educational and informative: the publishing of scientific articles (original research, case studies, and review articles) and other technical information, as well as editorials, letters, special articles (evaluations, position papers, reviews of nonscientific subjects),

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2. Ropes MW. Characteristics, manifestations, and pathologic findings. In: Ropes MD, ed. *Systemic Lupus Erythematosus*. Cambridge, MA: Harvard University Press. 1976; 50-4.

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DOCTORS & DISCIPLINE

The opinions expressed in this article are those of Dr. Winchell and have not been endorsed by the BPQA.

Comment by Dr. Cheryl Winchell, a member of BPQA.

Doctor or drug peddler?

The Board of Physician Quality Assurance (BPQA) received a call from the Drug Enforcement Agency (DEA) about a physician arrested for diverting narcotic drug prescriptions for non-medical uses. Board staff called the members of the BPQA and gave an outline of the information obtained from the DEA. Board members were told that the doctor had been under surveillance for some time. On several occasions, a trained agent had presented himself as a patient to Dr. X and essentially requested narcotics. The agent didn't allege that he was ill or in pain, just that he wanted drugs. In fact, he said he had a friend who would like some too. Did the doctor oblige? For the cost of an office visit, the agent was provided with prescriptions for Valium and Percocet. The doctor advised the agent that it would be best to fill the prescriptions at a pharmacy a good distance from his office so he wouldn't be questioned. The "office visit" took about three minutes. The doctor expected to be paid in cash. No employees worked for Dr. X. He accepted the cash payment from the agent and put the money in a drawer. Subsequently, numerous persons were seen coming and going to Dr. X's office. Their visits seemed to take only a few minutes.

The DEA visited pharmacies near Dr. X's office. They asked for a printout of Dr. X's narcotic prescriptions and the local pharmacies complied. (The agent feared he would get a hernia carrying the printouts and may actually need his Percocet.) Board members were queried whether, based on this information, they would feel comfortable voting for an emergency suspension of Dr. X's license. He had been released by the court on his own recognition. It takes eight votes to charge a physician with a breach of the Medical Practice Act. After eight phone calls, the necessary votes had been cast. An emergency order notifying of the BPQA's intent to suspend his license was served on Dr. X. He was given the opportunity to appear before the BPQA within 10 days to explain why his license should not be suspended. Dr. X did not request a hearing. In fact, Dr. X seemed to have disappeared. All pharmacists in the state were alerted that Dr. X's license was suspended and that he couldn't legally practice medicine in Maryland. Subsequently, Dr. X's license was revoked after a hearing conducted before an Administrative Law Judge and confirmation by the BPQA. Dr. X did not make an appearance and no one was present to rebut the testimony offered by the prosecuting attorney.

This is a "no brainer" case. The BPQA's course of action is clear from the outset. Dr. X is not practicing medicine; he is peddling controlled drugs for the price of an office visit. The agent who visited the "doctor's office" related that Dr. X didn't even own a blood pressure cuff and he didn't keep medical records on his "patients." It doesn't get any easier than this to vote to take away a doctor's license to practice medicine. Although none of us on the BPQA ever had the pleasure of meeting Dr. X or hearing his side of the story, it is unlikely that any of us would forget this egregious case should Dr. X decide he would like his license reinstated. Perhaps Dr. X is in jail. Perhaps he has left the country. Good riddance, either way.

It seems Dr. X was originally trained as a surgeon, but decided that he wanted to be a general practitioner. In a short time he had a rapidly growing practice. I don't know how long he was providing "primary care" before the DEA and BPQA put him out of business. But the sudden change from a surgical specialty, which involves maintaining hospital privileges, to providing primary care, which does not necessitate hospital privileges, is a red flag that something questionable may be going on.

Sometimes a surgeon is seeing a decline in his or her referrals and feels doing some primary care is a good way to supplement a falling income. Other times the doctor has lost all his hospital privileges and becomes an overnight general practitioner. Rusty diagnostic skills and out-of-date treatments may be the result if the physician doesn't re-

DOCTORS & DISCIPLINE

fresh his or her skills before embarking on such a major career shift. But the physician in this position, often edged out of the hospital rather than officially sanctioned, may not have the insight to recognize his or her own limitations. Further, financial pressures and the inability to maintain one's former life style may lead some physicians to cheat. This can be as gross as Dr. X's approach or more subtle. Upcoding office visits, bringing people back for unnecessary follow-up visits, selling unproven remedies out of the office, and providing nontraditional treatments are often used as ways to replace lost income. The quality of the medical care given in such circumstances may be substandard, but it is rarely so glaringly poor that the BPQA gets a "slam/dunk" like Dr. X provided. And be-

cause the BPQA is complaint driven, we have no authority to investigate doctors who are likely to be providing substandard care in the absence of a complaint.

The BPQA is receiving an increasing number of complaints from insurers who are analyzing utilization patterns. They send their most egregious outliers to us for investigation. A typical example would be a therapist who charges for more hours of therapy than there are hours in the week (that's right, more than 168 hours of therapy). The private insurers look to the BPQA to stop such fraud. But those who defraud Medicare and Medicaid can look forward to huge fines and jail terms because they are subject to state and federal laws. All the BPQA can do is take away a physician's license if he or she defrauds private insurers, but I guess that's a start. Generally, physicians who have a license revoked can apply for relicensure after a year. Often, we relicense them. Increasingly, I have been asking myself, "Why?" ■

Med Chi Bicentennial Celebrations

Med Chi has already begun planning celebration activities for its bicentennial in 1999.

If you have ideas or suggestions, please call Margaret Burri at 410-539-0872 or 1-800-492-1056.

What Your Patients MAY BE READING

- **Amazing Medical Breakthroughs.**
New Hope for Cancer, Heart Disease, more.
Family Circle, September 7, 1997
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A flood of new minerals, herbs, and hormones promise to prevent every ailment from wrinkles to cancer. Which work, which hurt?
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One woman's story of what it's like to go off America's favorite crutch.
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From the latest research, the 10 best things you can do to safeguard your child's life and well-being.
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Anything from stress to colds can throw off your cycle. But sometimes the cause is more serious. Find out when you need to see a doctor.
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- **"Wake-up Call" on Ultrasound.**
A study shows that the test is three times more accurate in Europe than in the U.S.—at a quarter of the cost.
Business Week, September 15, 1997
- **Having Your Baby: When you can't take the pain.**
A description of your options, which you should discuss with your provider before going into labor.
Black Child, Fall 1997
- **Shopping for a Doctor.**
Asking a lot of questions is the key. If a doctor doesn't answer your questions, consider getting another doctor. Here's why and the questions you should ask.
The Sandwich Generation, Summer 1997 ■

Books, Etc.

Book review editor:
Chris Papadopoulos, M.D.

Managing Your Child's Crohn's Disease or Ulcerative Colitis. K. J. Benkov and H.S. Winter. New York: Mastermedia Limited. \$21.95 (hardcover).

Sponsored by the Colitis Foundation of America and written by two experienced pediatricians, coupled with multiple contributions from the Pediatric Affairs Committee of the Foundation, this book is a significant resource for the treatment of children with inflammatory intestinal disease. The diagnosis is only the opening step to therapy inasmuch as the practical and psychological aspects of these illnesses demand their requisite care.

The paramount principle of the guide is that if all concerned have a better understanding of what the illness does to the patient, parents and children could cope better with these problems and the protean manifestation of these diseases. This tri-

partite book includes sections devoted to diagnosis, treatment, and living with ileitis and colitis. In turn, drug and non-drug therapy, surgery, diet and nutrition, school and social issues, and hospitalization are discussed. A glossary of medical terms makes the reading much easier for the uninitiated. Unfortunately, the book does not have an index but this is the only negative feature in a wealth of lucid instructive material. The book is heartily recommended for parents of children with these diseases and for physicians interested in this topic.

JOSEPH M. MILLER, M.D.

Dr. Miller is a retired surgeon residing in Timonium, Maryland. ■

The Living Heart Brand Name Shopper's Guide. 3rd Edition. M.E. DeBakey, A.M. Gotto, Jr., and J.P. Foreyt with M.C. McMann and S. Jaax. Edited by S. Simpson. New York: Mastermedia Ltd. 280 pages. \$14.95 (softcover).

This book was written by medical and dietary professionals to provide comprehensive and easy-to-read information for lay individuals needing dietary guidance for any number of medical conditions. The model does not ask for unrealistic commitment of knowledge or time. Specific diets may be easily followed.

The authors have furnished diets to delay, reduce in intensity, or prevent certain diseases, particularly those involving older age groups. When indicated, patients may use the diets to lower serum cholesterol and triglyceride levels, control weight, decrease blood pressure, manage diabetes mellitus, or reduce the risk of colon cancer.

Undesirable substances may be eliminated from diets during the preparation of meals. The authors suggest a number of cooking tips to help solve these problems. For example, removing all excess fat from meat and poultry and cooking in small amounts of olive oil to decrease the fat content of a meal.

Foods may be selected at home before going to the supermarket. A prepared list

will save roaming the aisles to compare similar brands.

This encyclopedic shopper's guide contains values for calories, fat, saturated fat, carbohydrate, sodium, and fiber for about 5000 foods. When several products have the same brand name, the brand designation is mentioned only once. Sections are divided in a manner similar to that found in the supermarkets. This division permits the reader easy access to desired information.

This guide is unique in many ways. It identifies brand names and generic foods that are good dietary choices. Values for food ingredients are consistent with recognized government health standards. An increased familiarity with major health concerns and American foods make this text a potential and unrecognized value in almost all homes and certainly in the offices of physicians interested in these health problems.

JOSEPH M. MILLER, M.D.

Dr. Miller is a retired surgeon residing in Timonium, Maryland. ■

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Syphilis, Cyclospora, Bat Rabies, Infection Control Network, and Conferences

Syphilis Cases Are Steadily Increasing in Baltimore

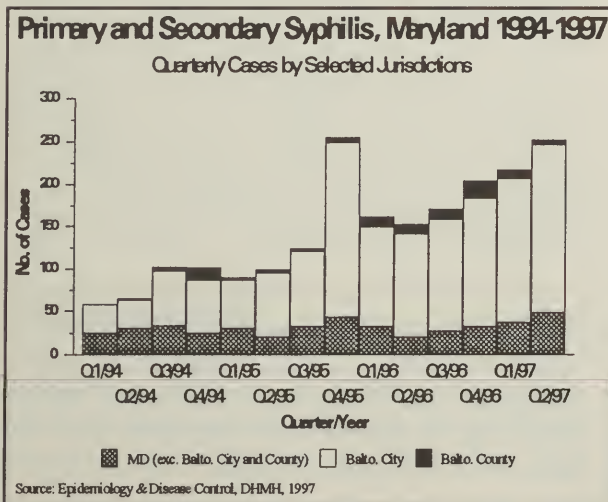
Infectious and congenital syphilis cases are steadily increasing in Baltimore City. In calendar year 1996 Baltimore City reported 557 cases of primary and secondary syphilis (i.e., "infectious syphilis"). This figure is 31% above the 427 cases reported in 1995, and 190% above the 192 cases reported in 1994 (see Figure, below). From 1989 through 1994, Baltimore City reported an average of 191 cases of infectious syphilis per year. One alarming result of the increase in adult cases has been an increased number of congenital syphilis cases. Recent data indicate that 37 cases of congenital syphilis have been reported for 1996--eight of these infants were stillborn. Baltimore City reported an average of 12 cases of congenital syphilis per year from 1993

through 1995. The number of primary, secondary, and congenital syphilis continues to increase in 1997. In the first six months of 1997, 369 cases of primary and secondary and 29 cases of congenital syphilis were reported in Baltimore City (Figure).

An increase in reported cases in Baltimore County was not seen until early 1996. Baltimore County reported 56 cases of primary and secondary syphilis in 1996, an increase of 269% from the 16 reported in 1995. Though relatively small in actual number, the magnitude of the increase is alarming. Congenital syphilis has increased as well. Five cases, of which two were stillborn, were reported for 1996 compared to no cases in 1995. In the first six months of 1997, Baltimore County reported approximately 30 cases of primary and secondary and two cases of congenital syphilis.

We urge every physician in the Baltimore Metropolitan Area to assist us in reversing these alarming trends. Early and appropriate screening, along with rapid diagnosis, treatment, and epidemiologic follow-up are crucial steps in successful syphilis control and congenital syphilis prevention.

✓**Know the symptoms of syphilis.** These include lesions in the urogenital or oral areas; generalized body rashes or rashes on palms, soles, or face; moist papular lesions in the urogenital or perianal area; lymphadenopathy; alopecia.



✓**Test symptomatic and high risk asymptomatic patients for syphilis.** Identified risks include a history of multiple sex partners, unprotected sex, infection with other STDs, and use of illicit drugs, especially crack cocaine.

✓**Test all pregnant women at the first prenatal visit and at 28 weeks of gestation** or the first prenatal visit after 28 weeks.

✓**Do a STAT serologic test for syphilis** on all women presenting in labor who have a history of unknown, spotty, or no prenatal care. Neither infant nor mother should be discharged before results are known.

✓**Treat** all diagnosed syphilis patients according to the *CDC 1993 Treatment Guidelines*.

✓**Report**, immediately, all cases of primary and secondary syphilis and all cases of syphilis in pregnant women to local health officials so that rapid follow-up may take place. Telephone reporting is encouraged during this epidemic.

✓**Consult** with the local health department on issues related to diagnosis, treatment, patient follow-up, and partner referral.

Obtain assistance and more information by contacting the STD Programs in Baltimore City (410-396-4448, fax 410-625-0688), Baltimore County (410-887-2713; fax 410-828-0986), or the Maryland Department of Health and Mental Hygiene (410-767-6688; fax 410-333-5529).

Cyclosporiasis in Maryland 1997

On July 2 and 3, 1997, the Alexandria Health Department (AHD) received two complaints of foodborne illness resulting from two seemingly unrelated catered events which both occurred on June 21, 1997. Upon further investigation, it was discovered that a food production plant based in Bethesda, MD had supplied the food to both events. Attendees of the two catered events were questioned by the AHD and ill individuals were asked to submit stool samples for the detection of enteric bacteria as well as ova and parasites. Stool results revealed that several attendees from both events were infected with *Cyclospora cayetanensis*, a newly recognized emerging pathogen which causes prolonged periods of severe diarrhea and extreme fatigue, as well as

gas and bloating. The food production plant implicated in these two outbreaks also supplied prepared food to other catered events as well as its food retail operation.

When data from the two initial outbreak investigations was analyzed, it was found that a pasta salad containing basil pesto sauce was the most likely vehicle of disease transmission for both catered events. Because recent experiences with *Cyclospora* outbreaks across the country involved fresh produce items, the ingredients of the basil pesto pasta salad were reviewed to determine the most likely source of *Cyclospora* contamination. It was found that the only fresh ingredient in the recipe likely to have been contaminated with *Cyclospora* was fresh basil. In response to this information, the food production plant voluntarily removed all basil containing products from both its catering operation and food retail chain.

On July 16, 1997, a joint meeting was called between the AHD, Virginia Health Department (VHD), Maryland Department of Health and Mental Hygiene (DHMH), Washington DC Health Department, and the Centers for Disease Control and Prevention (CDC), as well as representatives of the food production plant. At this time representatives of the food production plant agreed to supply a list of all catered events occurring after June 11, 1997. Customers who received catered food orders were contacted and asked if they or other attendees of these events had any recent gastrointestinal illness. Also, a press release was issued by the AHD, VHD, and DHMH alerting the public of the symptoms of *Cyclospora* and the risk of transmission from food purchased at or catered by the food production plant. Additionally, the public was asked to report *Cyclospora*-like illness regardless of their exposure to food from the implicated production plant. As a result, reports of illness were received from both the survey of catering customers as well as the general public.

A total of 63 clusters of illness were recognized in the Maryland/Virginia/ Washington DC metro area. Of these clusters, 31 had at least one laboratory confirmed case of *Cyclospora* infection, and the remaining 32 clusters were suspect clusters with cases having symptoms consistent with *Cyclospora*

related illness. In total, for the entire metro area, there were 310 reported cases of *Cyclospora*-like illness resulting from these clusters. Of these cases, 73 (24%) were lab confirmed. Eleven (35%) of the 31 lab confirmed *Cyclospora* clusters and 6 (19%) of the 32 suspect clusters of *Cyclospora*-like illness occurred in Maryland; 10 lab confirmed clusters and 6 suspect clusters occurred in Montgomery County, Maryland. In addition, six sporadic cases of lab confirmed *Cyclospora* infection were also reported.

No other retail food chain, catering service, or grocery store/supplier has been implicated in any of the reported *Cyclospora* clusters despite review of shipping records that indicates that the basil supplier for the food production plant also supplied basil to other area establishments. Similarly, basil from different shipments was implicated in these clusters. It is therefore believed that an infected food handler or multiple food handlers could have played a role in this outbreak of *Cyclospora* related illness. Similarly, improper food handling practices may also have contributed to this outbreak. In response to these findings, the food production plant is taking multiple steps, under the guidance of the DHMH Office of Food Protection and Consumer Safety, to reduce the possibility of a similar occurrence of illness in the future. This outbreak of *Cyclospora* infection marks the first instance in which improper food handling is believed to have contributed to the transmission of this disease.

In addition to the above clusters, Maryland experienced two small outbreaks of *Cyclospora* infection associated with Guatemalan raspberries in May of 1997. These two outbreaks resulted in a total of 10 ill and 5 lab confirmed cases. Also, an additional 5 sporadic cases have a reported history of raspberry consumption. Importation of Guatemalan raspberries was stopped on May 28, 1997. CDC officials are still investigating the source of *Cyclospora* contamination in Guatemala.

Rabies in Bats

In Maryland, there are ten species of bats, all of which are insectivorous. Bats are the only flying mammals and an average of eleven have been

confirmed as positive for rabies each year by the Maryland DHMH Laboratories Administration in Baltimore. The Centers for Disease Control and Prevention (CDC) have reported that bat rabies is enzootic in the contiguous United States, however, the reduction of bat populations is not a feasible or desirable strategy for rabies control in this reservoir. Bats are normally seen either flying or in a vertical position. When in a horizontal position, they are more likely to have rabies and should not be handled.

Insectivorous bats have small mouths only a few millimeters in size. As such, a bat bite may go undetected or unrecognized by a person who is bitten. Recent incidents of humans who have died of rabies do not include a history of a bat encounter. However, on laboratory examination, bat variants of rabies virus have been identified in these humans.

It is, therefore, most important to conduct a risk assessment in all situations involving potential human-bat contact. If at all possible, the bat should be captured and submitted for rabies testing. The CDC recommends Rabies Post-Exposure Prophylaxis (PEP) for all persons with bite, scratch, or mucous membrane exposure to a bat, if the bat is unavailable for testing. Additionally, in those situations in which there is a reasonable probability that contact with a bat has occurred, such as, a person sleeping in a room where a bat is found, or a bat in a room with a child, mentally disabled person or intoxicated person, PEP is appropriate.

Many individuals have heard of the possibility of airborne exposure to rabies from bats. To date, airborne rabies has been confirmed only in those persons who have been in caves where there are thousands of bats clustered together with a rain of urine, saliva, and feces. A single bat in a room has not been confirmed to spread rabies by the airborne route.

The Maryland Department of Natural Resources (DNR) teaches that insectivorous bats are needed to control the insect populations in the State. Even so, we must minimize human and animal contacts with bats by physically excluding them from houses and surrounding structures through the sealing of potential entrances. This is best accomplished when

bats that are in houses leave at dusk to feed. Further, they should never be handled by the public or kept as pets.

For more information, call the Center for Veterinary Public Health, Epidemiology and Disease Control Program at 410-767-6712.

Maryland Infection Control Network

The Maryland Infection Control Network (MICN) is an information network that has recently been established to assist in the **prevention and control of nosocomial infection** through:

- Consultation
- Training
- Communication
- Research.

The MICN is a joint venture between the Department of Health and Mental Hygiene, the University of Maryland and the Johns Hopkins University. Its purpose is to be a consortium of knowledge on infection control issues. For consultation, please call toll free 1-888-258-8989 or e-mail infect@welchlink.welch.jhu.edu.

Video Conferences

Hepatitis C: Diagnosis, Clinical Management, Prevention, a live interactive videoconference, co-sponsored by the Hepatitis Foundation International and the Centers for Disease Control and Prevention will be presented on Saturday, November 22, 1997 from 8:30AM-11:00 AM. The conference will focus on the latest developments in the epidemiology, diagnosis, clinical management, and prevention of hepatitis C. Two satellite downlink sites are available: St. Mary's Hospital in Leonardtown and the University of Maryland at Baltimore. Other sites may be available. The registration fee is \$25 per attendee, which includes continuing education credits. To register, call toll free, 1-(888)-CDC-FAXX and request document #130010 to have registration materials faxed back to you. If you have any other questions, please call the Center for Immunization, Epidemiology and Disease Control Program at (410) 767-6679.

Surveillance of Vaccine Preventable Diseases:

A live interactive satellite broadcast will be presented on December 4, 1997 from 12:00 Noon - 3:30 PM. This video conference will provide guidelines for vaccine-preventable disease (VPD) surveillance, case investigation, and outbreak control. The target audience is physicians, nurses, epidemiologists, infection control practitioners, laboratorians, and others involved in surveillance and reporting of VPDs.

Participants who pass an examination and complete a course evaluation can receive CMEs, CEUs, and nursing contact hours, pending approval of the course for accreditation. There is no fee for the course.

The Center for Immunization, Epidemiology and Disease Control Program will be providing several sites to view this videoconference. To request more information or registration materials, call (410) 767-6679.

Caring for Adolescents with STDs: A live interactive satellite broadcast for providers who treat STDs will be presented on Thursday, October 1, 1997 sponsored by the National Network of STD/HIV Prevention. Topics include: important public health issues; the biological and psychosocial aspects of adolescent development and its influence on sexuality and behavior; how to perform a comprehensive and productive interview and sexual history; effective communication and management techniques; and unique aspects of the physical exam and laboratory testing issues. Please call Jeannie Hoover at 410-396-3876 for registration information and a packet of down link sites at locations near to you.

HIV Prevention Update on Guidelines for Prevention Case Management and Partner Notification. A public health training network satellite broadcast HIV Prevention Update on Guidelines for Prevention Case Management and Partner Notification is scheduled for Thursday, October 23, 1997 from 1:00 - 3:30 PM EDT. For more information please call: 410-328-8639. Fax registration, call 410-328-9106.

The Johns Hopkins Medical Institutions

All courses at the Thomas B. Turner Building unless otherwise indicated. For information on continuing medical education activities, contact the Office of Continuing Medical Education, 720 Rutland Ave., Baltimore, MD 21205, 410-955-2959, Fax 410-955-0807 (e-mail: cmenet@som.adm.jhu.edu).

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|---|-----------------------|
| Care of aging adult persons with mental retardation: Emphasis on Down syndrome , sponsored by the Johns Hopkins Medical Institutions, at Renaissance Harborplace Hotel, Baltimore. Credits: 6.5 Cat 1 AMA credits. Fee: \$95/physicians; \$75/residents and nurses; \$35/DDA agency employees, social workers, counselors. | Oct. 20 |
| Advanced pediatric life support courses , sponsored by the Johns Hopkins University School of Medicine and the Johns Hopkins Pediatric Trauma Center. Credits: 21 Cat 1 AMA credits. Fee: \$595. | Oct. 27-29 |
| Computed body tomography for the technologist with CT registry review , sponsored by the Johns Hopkins University School of Medicine Body CT, department of radiology, at Peabody Orlando Hotel, Orlando, Florida. Credits: 20.5 Cat 1 AMA credits. Fee: \$475/technologists. | Oct. 30-Nov. 2 |
| Progress in practical pediatrics , sponsored by the Johns Hopkins University School of Medicine and the Children's Medical and Surgical Center. Credits: 10.5 Cat 1 AMA credits. Fee: \$165/physicians; \$110/residents, fellows, allied health professionals. | Nov. 7-8 |
| Type II diabetes: Current concepts in management , sponsored by the Johns Hopkins University School of Medicine. Credits: 7.5 Cat 1 AMA credits. Fee: \$50/physicians; \$35/residents, fellows, allied health professionals. | Nov. 15 |
| Women's health conference , sponsored by the Johns Hopkins Medical Institutions, department of gynecology and obstetrics, at Sheraton Baltimore North Hotel, Towson. Credits: 7 Cat 1 AMA credits. Fee: \$95/physicians; \$75/residents, fellows, allied health professionals. | Nov. 21 |
| Investigating and organizing clinical research , sponsored by the Johns Hopkins Medical Institutions, department of medicine. Credits: 8 Cat 1 AMA credits. Fee: \$250/physicians; \$195/residents, fellows, allied health professionals. | Nov. 21 |
| Eighth annual neurology for the primary practitioner , sponsored by the Johns Hopkins Medical Institutions, department of neurology, at Harbor Court Hotel, Baltimore. Fee: \$140/physicians; \$90/residents, fellows, allied health professionals. | Dec. 6 |
| Topics in ambulatory medicine , sponsored by the Johns Hopkins University School of Medicine and the Johns Hopkins Bayview Medical Center, at the Renaissance Harborplace Hotel, Baltimore. Fee: \$550/physicians; \$325/residents, fellows, allied health professionals. | Dec. 10-12 |

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.

The Johns Hopkins Medical Institutions (continued)

Johns Hopkins sports medicine grand rounds. Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Advances in laser applications for ophthalmology, Long Beach, California. Info: Mary A. Johnson, Ph.D., University of Maryland at Baltimore, 22 S. Greene Street, Baltimore, MD 21201. 410-328-5930, FAX 410-328-6346. **Oct. 12**

Self-Directed CME Activities

CD-ROM-based interactive multimedia radiology teaching file for Mac or PC w/single-user licenses (SUL), site licenses (SL), or multisite licenses (MSL). Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Cancer prevention in community practice, sponsored by the Medical and Chirurgical Faculty of Maryland, at Washington County Hospital Association, Washington County. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Oct. 8**

24th annual recent advances in clinical medicine, sponsored by The University of Virginia Office of CME, Omni Charlottesville Hotel, Charlottesville, Virginia. Credits: 18 Cat 1 AMA credits. Fee: \$375. Info: 804-924-5318, Fax 804-982-1415 (e-mail: lem8a@virginia.edu). **Oct. 15-17**

Infectious disease '97 board review: A comprehensive review for board preparation, sponsored by The Center for Bio-Medical Communication, at the Ritz-Carlton, Tysons Corner, McLean, Virginia. Credits: 40 Cat 1 AMA credits. Fee: \$895/physicians; \$695/physicians-in-training. Info: 201-385-8080, Fax 201-385-5650 (e-mail: cbcbiomed@aol.com). **Oct. 15-19**

Neuroradiology Update, sponsored by The University California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, California. Credits: 13 Cat 1 AMA credits. Fee: \$300/physicians; \$200/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Oct. 16-17**

New techniques in urinary incontinence and female urology, sponsored by The Washington University School of Medicine, at the Eric P. Newman Education Center, Washington University Medical Center, St. Louis, Missouri. Credits: 8.5 Cat 1 AMA credits. Fee: \$200/physicians; \$100/physicians-in-training. Info: 800-325-9862, Fax 314-362-1087. **Oct. 18**

Miscellaneous (continued)

- UCSD postgraduate radiology course: Musculoskeletal, obstetrical, and body imaging, neuroradiology, chest imaging, and women's imaging and pediatrics**, sponsored by The University California, San Diego, School of Medicine, at the Hotel Del Coronado, Coronado, California. Credits: 40 Cat 1 AMA credits. Fee: \$1,000/physicians; \$700/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Oct. 20-24**
- Musculoskeletal MR**, sponsored by The University California, San Diego, School of Medicine, at the Westin Resort Hotel, Hilton Head, South Carolina. Credits: 20 Cat 1 AMA credits. Fee: \$550/physicians; \$375/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Oct. 22-26**
- New techniques and concepts in cardiology**, sponsored by The American College of Cardiology, at the Hyatt Regency Capitol Hill, Washington, D.C. Credits: 16 Cat 1 AMA credits. Info: 800-253-4636, ext. 695, Fax 301-897-9745. **Oct. 23-25**
- Sleep disorders update**, sponsored by The Washington University Multidisciplinary Sleep Medicine Center and the Office of Continuing Medical Education, at Eric P. Newman Education Center, Washington University Medical Center, St. Louis, Missouri. Credits: 6 Cat 1 AMA credits. Info: 800-325-9862, Fax 314-362-1087 (e-mail: WUCME@msnotes.wustl.edu). **Oct. 25**
- Occupational and environmental medicine: Clinical practice in progress**, sponsored by the American College of Occupational and Environmental Medicine, at the Opryland Hotel in Nashville, Tennessee. Info: 847-228-6850, ext. 184, Fax 847-228-1856. **Oct. 26-30**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Memorial Hospital at Easton, Talbot County. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Oct. 29**
- Surfing the legal net**, sponsored by Consult, Inc. at Marriott Metro Center, Washington, DC. Info: 703-685-0035, Fax 703-271-0980. **Nov. 1**
- Breast imaging and interventions: A multidiscipline approach**, sponsored by The American Association of Physician Specialists and The International Institute for Continuing Medical Education, at The Ritz Carlton Resort Hotel, Naples, Florida. Credits: 28 Cat 1 AMA credits. Fee: \$695/physicians; \$495/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Nov. 3-6**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at North Arundel Hospital. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Nov. 20**
- Fall Regional Conference, Infectious disease update**, sponsored by The Maryland Academy of Family Physicians (MAFP), at Harbortowne Golf Resort & Conference Center, St. Michaels, Maryland. Credits: 6.25 AAFP Prescribed credits. Fee: \$75/MAFP members; \$100/nonmembers; \$50/allied health professionals. Info: 410-747-1980. **Nov. 8**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Prince George's Hospital Center. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Dec. 3**
- Surveillance of vaccine-preventable diseases**, presented via satellite by the Centers for Disease Control and Prevention, hosted by the Center for Immunization, Maryland Department of Health and Mental Hygiene. Credits: CMEs available. Fee: none. Info: Sandra Kash, 410-767-6679. **Dec. 4**

Miscellaneous (continued)

- Rural Health Conference, Communities leading the way**, sponsored by The Maryland Academy of Family Physicians (MAFP), at Loews Annapolis Hotel, Annapolis, Maryland. Credits: 17 AAFP Prescribed credits. Fee: \$125. Info: 410-747-1980. **Dec. 4-6**
- 25th annual Williamsburg conference on heart disease**, sponsored by the American College of Cardiology at Williamsburg Lodge, Williamsburg, Virginia. Credits: 19 Cat 1 AMA. Info: 800-253-4636, ext. 695, Fax 301-897-9745. **Dec. 7-10**
- 14th annual CME clinical update in pulmonary medicine**, sponsored by the department of pulmonary medicine, Deborah Heart & Lung Center, Browns Mills, NJ, at the Trump World's Fair Casino, Atlantic City, New Jersey. Credits: 7 Cat 1 AMA. Fee: \$175/physicians; \$100/allied health professionals, physicians-in-training (until Oct. 14); \$225 and \$130, respectively, after Oct. 14. Info: 201-385-8080, Fax 201-385-5650 (e-mail: jrosenberg@cbcbiomed.com). **Dec. 13**

Continuously throughout the year

- Fluorescein angiography conference**, sponsored by the Retina Center of Maryland, Baltimore. First and third Mondays of each month; 8:00 a.m. - 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.
- Sinai Hospital of Baltimore medical grand rounds**, Zamoiski Auditorium, Thursdays, 9:00 a.m. - 10:00 a.m. Info: 410-578-5528.



PHYSICIAN'S RECOGNITION AWARD

During June and July 1997, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

Bernard James Arseneau
Sumitra L. Bawa-Parekh
Shakti Bhatnagar
Antonio Sergio Cassanego
Frank Edgar Chang
Kailash Chopra
Joseph John Colella
Constantino Costarigos
Joseph R. Cowen
Ronald Lee Diener
Thomas Edward Dooley
Michael Anthony Ellis
Najla Nouri Falaki
Eric Finzi
Robert Lawrence Flynn
Edmund John Forte
James Patrick Jarboe

Murray Alvin Kalish
Martin Zelig Kanner
Sheppard Kaplow
Antoine Elia Kfuri
Richard Jay Kolker
Lawrence Scott Linder
Garrett John Lynch
Murli Narain Mathur
Sherman Alfred McCall
Gurbux H. Nachnani
Richard Jos Nasca
John Francis O'Neill
Lawrence Chas Pakula
Elma G. Panizales
Estella C. Parrott
S. Pathmanathan
Frederick W. Plugge

Manoochehr Pooya
John Manfred Ratino
James William Rice
Samuel Beryl Ritter
Alejandro Javier Rivera
Daniel Robert Rodgers
Erik Samuel Robinson
Edward F. Sanford
Melvin Silverman
Jeffrey L. Soulen
Myron James Szczukowski
Jean W.A. Temeck
Alan Scott Tuckman
Christopher D. Turner
James Vorosmarti
James Christopher Wasson
Stanley Irving Wolf

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- Cost for nonmembers is \$8.00 per 35 characters or portion thereof, with a minimum cost of \$50.00 per advertisement.
- Advertisements placed for the benefit of an HMO or a hospital are charged the nonmember rate.
- Advertisements for services (e.g., collection agencies) or for items not related to the practice of medicine (e.g., real estate sales) are limited to a maximum of three placements per calendar year.
- Spouses of deceased members are entitled to two complimentary insertions for the disposal of the deceased physician's practice or equipment.
- Box numbers are provided free of charge.
- Advertising copy and payment (checks should be made out to *Maryland Medical Journal*) should be sent seven weeks prior to the first of the month in which the advertisement is to appear.

Box replies, advertising copy, and prepayments should be sent to:

Vivian Smith, *MMJ*, 1211 Cathedral St.,
Baltimore, MD 21201-5585

*For more information, call Vivian Smith at
410-539-0872 or 1-800-492-1056, ext 308.*

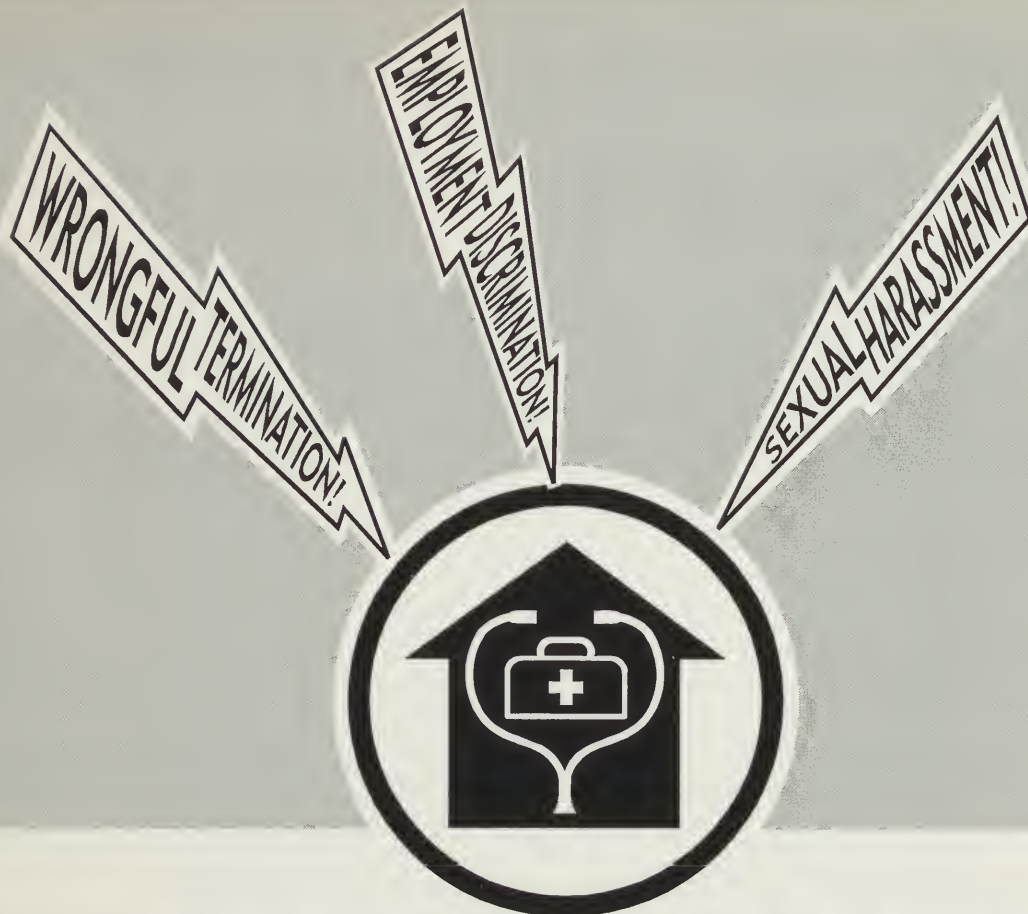
PHYSICIAN PLACEMENT SERVICE

The Medical and Chirurgical Faculty of Maryland maintains a Placement Service for the convenience of Maryland physicians, hospitals, and communities in search of candidates for positions available in our state. A detailed description of such opportunities should be forwarded to:

Physician Placement Service
1211 Cathedral Street
Baltimore, MD 21201
410-539-0872
1-800-492-1056

Physicians wishing to locate in Maryland are invited to submit a résumé to be kept on file with the Physician Placement Service. Candidates are requested to inform the Faculty when they are no longer available for consideration opportunities in Maryland.

MMJ announcements for physician placements in the classified advertisements are charged at the regular classified advertising rate.



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—515—

*Diagnosis of Pfiesteria-human
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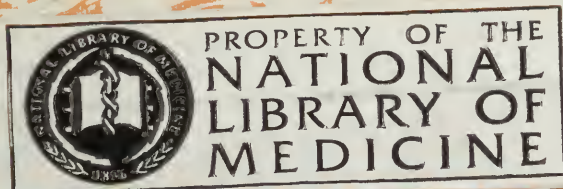
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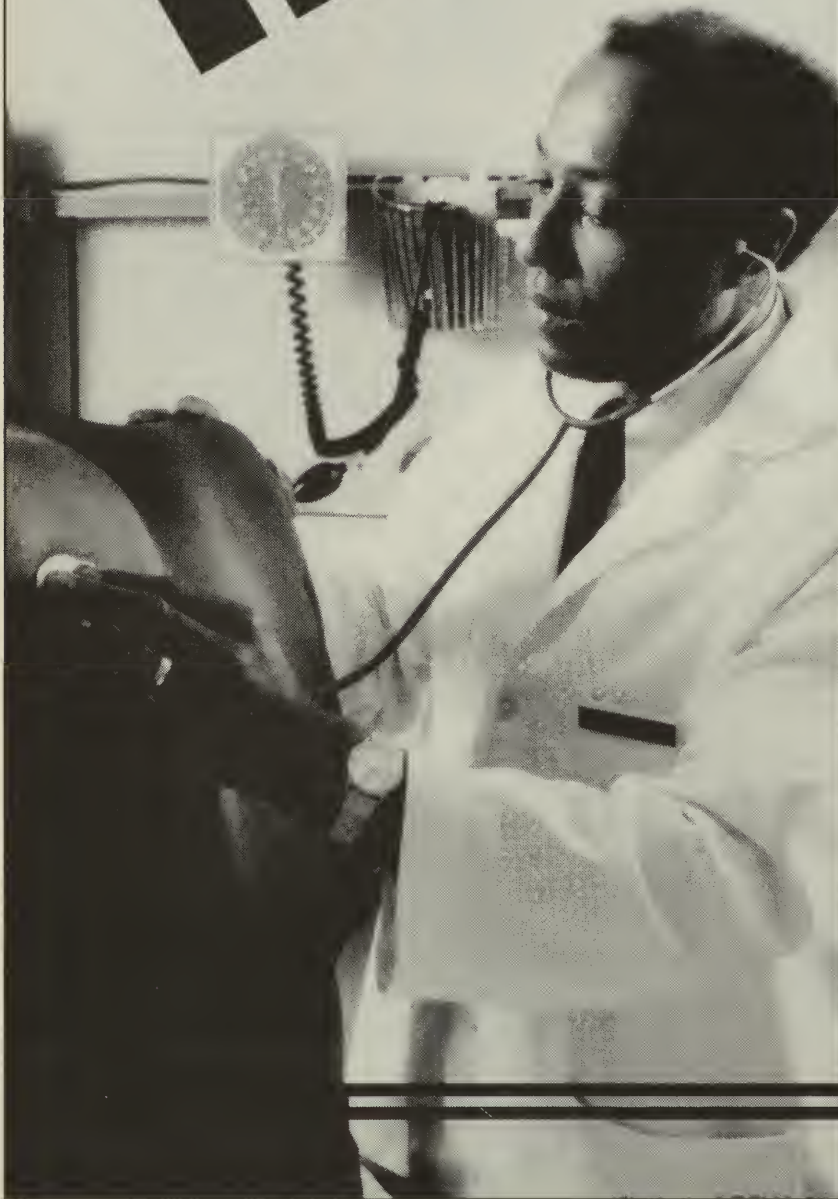
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Editor's Notes

■ *PFIESTERIA* OUTBREAK

As all our readers must be aware, Maryland is the center of a new and confusing controversy that appears related to pollution of waterways, the killing of many fish in certain streams and rivers, and even the infection of some Marylanders who have been exposed to these waters and their vapors.

In this issue we are presenting an article by Diane L. Matusak, M.D., M.P.H., associate director, community and public health administration, and others from the Maryland Department of Health and Mental Hygiene, including the secretary. It is a cogent, straightforward review of the problem from its known beginning to our present state of knowledge.

We are also publishing a report of five patients presumed to have been infected who were seen and treated by Ritchie Shoemaker, M.D., of Pocomoke City, Maryland. Dr. Shoemaker has since reported additional cases to the medical team.

It is recognized that much is yet to be determined and that some "facts" believed to be true today may have to be reconsidered. However, we feel the physicians of Maryland should be alerted and kept abreast of what is happening and of the current thinking through their official medical journal rather than via newspapers and magazines.

As the story unfolds we shall make every attempt to bring it to you. Stay tuned.

■ SUPPLEMENT ON CHEST PAIN UNITS

The reader's attention is drawn to our supplement that was edited by Raymond D. Bahr, M.D., a cardiologist and the medical director of the Paul Dudley White Coronary Care System at St. Agnes HealthCare, Baltimore, Maryland. This supplement, *The Strategy of the Chest Pain Units (in Emergency Departments) in the War Against Heart Attacks*, contains the proceedings from the First Maryland Chest Pain Center Research Conference, held at St. Agnes.

In 1981 at St. Agnes, Dr. Bahr established the Chest Pain Emergency Department (CPED), the first early cardiac care center in the world. The primary purpose of the CPED is the prompt, effective treatment of patients presenting with heart attack/sudden death. The CPED is coupled with an aggressive education program that teaches the community the early warning signs of a heart attack.

Since its inception, the CPED has gone through a second generation of early heart attack care by becoming a cardiac intervention area, with a fast track for early thrombolytic therapy for myocardial infarction patients. Presently, the CPED is in its third generation of early heart attack care, which encourages early hospital entry for patients with prodromal angina and teaches people in the community to be early cardiac care givers and help victims by getting them to the hospital while symptoms are minimal and denial is maximal.

The concept, now in its 16th year, has spread widely to more than 700 hospitals in the United States and around the world. It has already saved countless lives and diminished crippling cardiac disease in those who have survived. Dr. Bahr's goal is further dissemination of this knowledge to all physicians, hospitals, care givers, and the laity. We are happy to be a part of this noble aspiration.

We are also pleased to include in this supplement remarks by Richard Horton, M.D., editor of the internationally prodigious British medical journal *Lancet*. He presents an interesting philosophical approach to medical and clinical practice research that he terms "interpretive medicine." This is what he notes the contributors to this supplement have attempted "with the goal to challenge other states as well as other countries to do similarly."

MARION FRIEDMAN, M.D.

Editor ■

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Reader finds journal useful

I want to take this opportunity to thank you for such an informative and absolutely excellent journal on medicine. It's much appreciated and used to good benefit. It is great to be kept abreast of the latest here

in Maryland, and the United States as well. Again, my thanks to you for fine journalism.

Yours truly,

GRACE V. LINDSAY, M.D.

Dr. Lindsay practices in Hyattsville, Maryland. ■

LETTERS TO THE EDITOR



The editorial board of the *Maryland Medical Journal* welcomes comments, criticisms, recommendations, and observations from all its readers. Please submit letters to: Editor, *Maryland Medical Journal*, 1211 Cathedral Street, Baltimore, MD 21201-5585

Direction of medical care one physician's view

Medicare and managed care operations are now being rapidly amalgamated to provide therapy for 300,000 Maryland patients. The impact upon patients and doctors is not known. It is certain that doctors will receive lower levels of compensation.

Twenty-first century medicine is rapidly being redesigned to engender a generalistic approach to disease that will operate on an out-patient basis. Managed care principles, with a heavy accent on the business problems of medicine, are directing attention away from providing good care.

The primary care program is expected to coordinate the entire gamut of therapy, both in and out of the hospital. Economic pressures by multiple health groups supplying insurance against the costs of illness have led to a multitude of varied working environments governed by numerous regulatory systems. It is apparent that the delivery of inadequate care may only result in a greater morbidity and mortality. Quality of care is being traded for economic gain.

Dedication, compassion, and philanthropy have long been considered essential to medical practice. The earliest priest-physicians served as advocates to the gods for their patients. Early direct medical care was later based on a Judeo-Christian moral philosophy subsequently supported by the Greco-Roman concept of logic.

With the advent of a more scientific approach in the nineteenth century, the use of the Golden Rule became more apparent. Now, such covenantal care is being replaced by a business relationship based upon a contract. Physicians are still seeing patients but they must comply with guidelines created by managed care. If these managed care companies provided good care at a better economic rate, the programs would be acceptable. Cost containment could be beneficial if offered without medical restriction. Cooperation between primary care providers and specialists might be enhanced with nothing but benefit to the patient.

In 1995, more than nine billion dollars were generated as profit from health care. This event occurred at a time when Congress and the public were deeply concerned with the costs of good therapy.

In the era of Hippocratic medicine (450 B.C.), doctors swore by Apollo that they would act in the best interests of the patient at all times. In addition, at the beginning of the Christian era, the precepts of "love thy neighbor" and "do unto others" provided a firm foundation for covenant treatment. A change from a covenant to a contract era would be an impediment to the delivery of high-quality therapy.

JOSEPH M. MILLER, M.D.

Dr. Miller is a retired surgeon who resides in Timonium, Maryland. ■



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Toxic *Pfiesteria* and human health

Diane L. Matuszak, M.D., M.P.H., Martin Sanders, Ph.D.,
Jean Lin Taylor, M.P.H., and Martin P. Wasserman, M.D., J.D.

From the [Maryland Department of Health and Mental Hygiene] where Dr. Matuszak is associate director [USA] community and public health administration, Dr. Sanders is an epidemiologist, division of outbreak investigation, Ms. Taylor is an epidemiologist, division of communicable disease surveillance, and Dr. Wasserman is Secretary.

ABSTRACT: Toxic activity of a *Pfiesteria*-like organism occurred for much of 1997 in the waters of the lower Pocomoke River on Maryland's Eastern Shore. Maryland's experience with these toxic blooms of dinoflagellates, current knowledge of their potential human health effects, and the actions taken by state government agencies in response to a potential public health threat are reviewed. A medical diagnostic team commissioned by the Department of Health and Mental Hygiene evaluated a group of persons with intense exposures to lesioned fish or the waters from which they came and/or prominent symptoms following exposure to affected waters or lesioned fish. The principal findings of the team included consistent complaints of memory problems, acute burning of the skin following direct contact with water, and respiratory irritation. Findings on examination were limited to neurocognitive deficits in short-term memory and learning difficulties. Physicians and citizens are asked to continue to report, through their local health departments, illnesses thought to be related to exposure to lesioned fish or the waters from which they are taken. Persons with questions or wishing to report finding lesioned fish should call the state *Pfiesteria* hotline at 1-888-584-3110.

Fish kills associated with skin ulceration and of unknown cause were reported as early as 1972 in Asia, reaching epidemic proportions in India before a 1991 study by Chattopadhyay et al.¹ This study isolated *Aeromonas hydrophilia* from the tissues of ulcerated fish leading to a diagnosis of

atypical infection with this common waterborne bacterium as a possible cause of what was termed ulcerative disease syndrome in fish.¹ In 1992, a newly identified toxic dinoflagellate, named *Pfiesteria piscicida* because it kills fish, was described by scientists at North Carolina State University, including JoAnn Burkholder and Edward Noga. This single-celled organism has been implicated in several large fish kills in the Pamlico Sound and the Neuse River there.² The dinoflagellate has multiple life stages: at least two dozen have been described, including cysts, vegetative cells with two flagella, and amoeboid forms.³ Several stages, including both flagellated and amoeboid forms, are toxic to all finfish challenged in the laboratory. The most toxic form is a small vegetative flagellated form that produces large amounts of exotoxin in the presence of living fish and is apparently stimulated by an unknown substance in fish excreta. Live finfish stimulate toxin production; live shellfish have not been demonstrated to do so in the laboratory.^{2,4}

Maryland's experience

Maryland's experience with the effects of toxic forms of *Pfiesteria* or *Pfiesteria*-like organisms began in October 1996 when watermen fishing on the Pocomoke River reported finding fish with unusual, ulcerative skin lesions. There was no evidence of a fish kill. Officials and scientists from the Maryland Department of Natural Resources (DNR) investigated and collected samples of water, sediment, and fish for analysis. Tests indicated that water quality conditions were within acceptable ranges. Water samples collected and sent to the laboratory of Dr. Burkholder in October 1996 were reported as negative for *P. piscicida* in January 1997.

When fishing resumed in the spring of 1997, watermen again noted fish with ulcerative lesions and several waterman reported feeling ill. They attributed their illnesses to exposure to the lesioned fish. A reporter from a Washington, D.C., television station reported the watermen's story and collected a water sample in early May 1997 that he sent to Dr. Burkholder's laboratory. The result of testing on that sample was returned as a preliminary positive for a *Pfiesteria*-like organism. DNR assembled an interagency team to investigate the problem on the Pocomoke River. In April and May, DNR sampled nearly 2,500 fish from the Pocomoke River and found that 0% to 10% had lesions. These proportions were not thought to be exceptional for Chesapeake Bay fish or to indicate serious problems for fish populations. Maryland had not experienced a large fish kill in several years. However, commercial fishermen were reporting higher

proportions of fish with lesions in near-shore nets and the situation warranted further investigation.

In response, DNR established a hotline to receive citizen reports of lesioned fish. The existing fish kill team and other teams were dispatched to investigate all reports of fish with lesions or unusual behavior patterns or fish kills. The Department of Health and Mental Hygiene (DHMH) established a surveillance system to look for human health effects. The local health officers in Somerset, Worcester, and Wicomico counties sent letters to all health care providers practicing in those counties and requested reports of any illnesses they thought could be related to exposures to fish with lesions or the waters from which they were taken.

From August 1 to 3, an interstate colloquium was held on the campus of Salisbury State University that brought together scientists and experts representing federal, state, (Maryland, Virginia, Delaware, Florida, North Carolina, and South Carolina) and academic institutions to discuss *Pfiesteria* and to review the state's action plan. At that meeting, a private physician informed us of a patient he was convinced had an illness caused by exposure to the Pocomoke River. We also heard statements from a panel of watermen about health problems that had arisen after they started catching fish with ulcerative lesions. DHMH made a commitment to commission

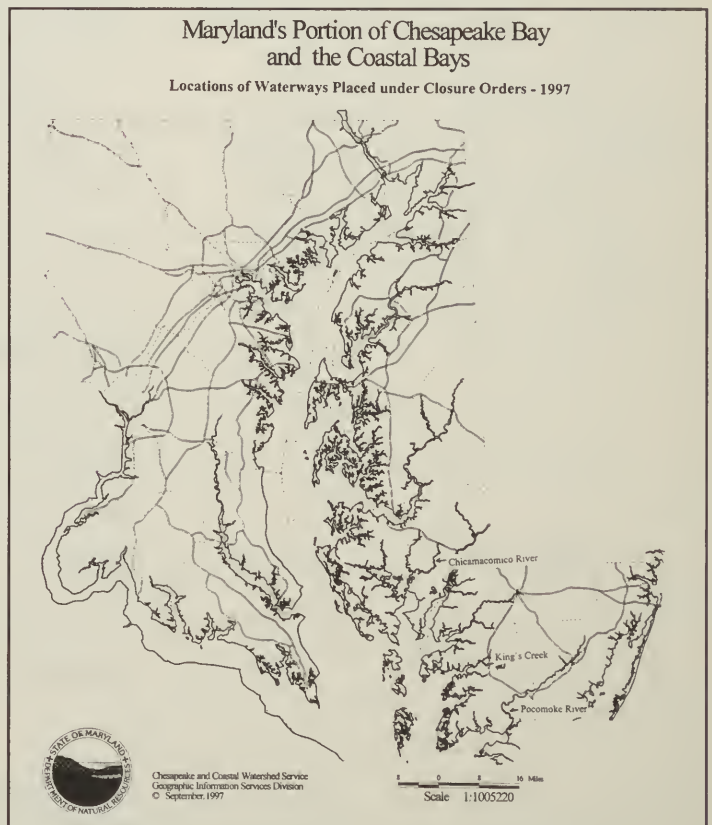


Figure 1. Location of waterways placed under closure orders—1997.

TABLE 1. Protocol for closing and reopening rivers affected by *Pfiesteria* or *Pfiesteria*-like events

A closure will be recommended when:

A significant fish kill in progress is confirmed and the affected fish are exhibiting sores consistent with toxic activity of *Pfiesteria* or *Pfiesteria*-like organisms

OR

A significant number of fish are confirmed to be acting erratically (and no other explanation for the behavior is apparent [e.g., low dissolved oxygen levels] or 20% or more of one species (from a minimum of 50 fish of the species collected) are exhibiting fresh sores consistent with toxic activity of *Pfiesteria* or *Pfiesteria*-like organisms or there is evidence of increased activity of *Pfiesteria* or *Pfiesteria*-like organisms, as reflected by an increase in the number of fish with lesions consistent with toxic activity of *Pfiesteria* or *Pfiesteria*-like organisms.

A reopening will be recommended when:

The conditions that initiated the closure have ceased for 14 days.

a medical diagnostic team consisting of experts from the University of Maryland and The Johns Hopkins University to evaluate a group of persons who reported intense exposures to fish with lesions or the waters from which they were taken or prominent symptoms and determine the possible nature and severity of any human health effects.

On August 6, an estimated 10,000 to 15,000 fish were discovered dead or dying on the lower Pocomoke River by local watermen and confirmed by DNR. State and local health officers issued a public health advisory warning the public to avoid contact with the affected area. When persons failed to comply with the advisory, it was upgraded to a public health closure order covering the affected area. The closure order was lifted on August 13, four days after the end of the fish kill. Preliminary results of water samples taken during the August 6 to 9 fish kill indicted the likely presence of toxic activity by a *Pfiesteria*-like organism. Dissolved oxygen levels at the time of the fish kill were high enough to rule out hypoxia as a primary factor in the fish kill.

The medical diagnostic team performed evaluations of the first group of persons with intense exposures or prominent symptoms on August 22. On August 26, a fish kill that began in the Virginia waters of the Pocomoke Sound occurred. The entire area of the lower Pocomoke River was again placed under a public health advisory. When the medical team issued its interim report on possible health effects on August 29, the affected area was placed under a closure order. A hotline was established working with the Maryland Infection Control

Network, which is cooperatively operated by The Johns Hopkins University and the University of Maryland, with Johns Hopkins University providing coverage to answer citizens' questions regarding health issues. Eventually, this hotline was merged with the hotline operated by DNR to allow the public to receive comprehensive information regarding *Pfiesteria* concerns from a single source. Subsequent closure orders were issued for King's Creek, on September 10, after an investigation of a hotline report confirmed the presence of a significant number of distressed fish, for the Chicamacomico River, on September 14, because of a confirmed fish kill. A protocol was developed to govern the closing and reopening of waterways for the protection of the public health. The protocol is summarized in Table 1.

Human health effects

Toxic dinoflagellates are known to cause a variety of human health problems. A summary of some of the known organisms, toxins, and health effects is shown in Table 2. Case reports of illnesses in laboratories thought to be due to exposures to aerosols of *P. piscicida* toxins were published in 1995 by Glasgow et al. Reported symptoms included tearing/eye irritation, weakness, respiratory problems, memory loss, joint pain, nausea, abdominal pain, emotional changes, skin lesions, paresthesia, headache, myalgia, vomiting, and excessive perspiration.^{3,5} An unpublished descriptive epidemiological study of persons exposed to three fish kills attributed to *P. piscicida* on the Pamlico River, Goose Creek, and the Neuse River in North Carolina was conducted in 1995 by Dr. Peter Morris, who was working for the North Carolina Department of Environment, Health, and Natural Resources. This study found no consistent pattern of symptoms among persons meeting exposure criteria. There was only one report of skin problems among seven persons exposed to a fish kill on the Pamlico River. Seven of 12 persons similarly exposed on Goose Creek reported any symptoms, six of these had symptoms consistent with exposure to hydrogen sulfide. A smell of hydrogen sulfide was noted during the episode. Fifteen of 32 persons exposed to a Neuse River fish kill reported at least one symptom; five reported persistent cognitive complaints. Three had cognitive testing approximately two months after exposure; two were found to have cognitive deficits but preexisting conditions or testing circumstances may have influenced the results.¹¹ A large analytical epi-

miological study of the health of crabbers in North Carolina is being conducted by a team at East Carolina University led by Dr. David Griffith. Approximately 250 crabbers who work on waters affected by *Pfiesteria* are being compared with two control groups: 114 crabbers working in areas unaffected by toxic *Pfiesteria* and 125 non-fishing community residents. Preliminary results issued in a press release in May 1997 show that both groups of crabbers report higher incidences of skin disorders than community controls, but crabbers working in areas with toxic *Pfiesteria* blooms had no more health problems than crabbers working in waters that had not had toxic *Pfiesteria* blooms. Final results of this study have not yet been published.

The surveillance system established by DHMH, which gathered information on both symptoms and exposures to lesioned fish or the waters in which they were found, facilitated the identification of a group of persons who

reported intense exposures and/or prominent symptoms. DHMH asked the medical diagnostic team assembled by our two universities to evaluate this group to delineate the nature and severity of possible health effects. The medical diagnostic team was led by J. Glenn Morris, Jr., M.D., M.P.H., and T.M., professor of medicine, University of Maryland School of Medicine. Other team members included, from The Johns Hopkins University School of Medicine, Patricia Charache, M.D., professor of pathology, medicine, and oncology, and Trish M. Perl, M.D., M.Sc., assistant professor of medicine, and from University of Maryland School of Medicine, Lynn M. Grattan, Ph.D., associate professor of neurology, Mark H. Lowitt, M.D., assistant professor of dermatology, and David Oldach, M.D., assistant professor of medicine.

The medical diagnostic team took a detailed history and performed a detailed physical examination. Each patient was

TABLE 2. Comparative symptoms of dinoflagellate toxins/organisms reported in North America*

Condition	Toxin	Organism	Symptoms	Incubation	Duration
Ciguatera fish poisoning	ciguatoxin	<i>Gambierdiscus toxicus</i>	diarrhea vomiting abdominal pain pain (lower extremities) weakness (lower extremities) paresthesias "temperature reversal" "aching teeth" coma (rare) death (rare)	1 hour – 2 days N/A N/A	weeks to months (symptoms may recur)
Paralytic shellfish poisoning	saxitoxin	<i>Gonyaulax tamarensis</i> <i>Alexandrium spp.</i>	paresthesias (mouth/extremities) diarrhea vomiting abdominal pain ataxia (rare) respiratory arrest (rare) death (rare)	minutes to hours N/A	2 – 3 days
Neurologic shellfish poisoning	several toxins	<i>Gymnodinium breve</i>	paresthesias "temperature reversal" diarrhea vomiting ataxia	minutes to hours	days (self-limiting)
Amnesic shellfish	domoic acid	<i>Nitzschia pungens</i> (renamed <i>Pseudo-nitzschia multiseries</i>)	gastroenteritis memory confusion, deficits short-term memory loss death	minutes to hours	chronic residual

*Adapted from references 6, 7, 8, 9 and 10

TABLE 3. Recommendations for managing concentration and memory difficulties

Lynn M. Grattan, PhD, 1997

- Plan in advance any multi-step activity and complete the activity one step at a time.
- Perform household or work-related tasks one at a time. Do not start a new activity until the one you are working on is finished.
- Use self-instruction several times a day. For example, ask yourself, "Am I being distracted?" "Am I wandering?" What should I be doing now?"
- It is recommended to use a timer or an alarm to help to focus attention for a specific period of time.
- A daily planner should be used with the day's events and activities listed in a step-wise fashion.
- Productivity and learning will be enhanced in a low-stimulus environment. For example, read, study, balance your checkbook, or work in areas free from distractions such as radio, television, or people engaged in conversations. Consider using ear plugs to block out excess noise. At school or work, attempt to sit in a quiet part of the room, away from friends, windows, doors, and other possible distractions.
- Organize work environment to eliminate distractions.
- Memory will be enhanced by rehearsing and repeating the information to be learned.
- Work when well rested and take frequent rest breaks if necessary.
- Allow enough time for adequate sleep.

There may be situations in which such evaluation is desirable. Specific treatment for the neurocognitive deficits is not available. To the extent possible, it is recommended that affected persons be removed from exposure. A variety of compensatory mechanisms, recommended by Dr. Grattan, for persons who are experiencing memory difficulties are detailed in Table 3.

Discussion

There is ample evidence that several Maryland waters were affected by toxic *Pfiesteria*-like dinoflagellates during the summer of 1997. Previously reported human health effects of other toxic dinoflagellates have been largely associated with ingestion of toxins. An exception is the exposure related to inhalation of aerosols of toxins *Gymnodinium breve*.¹³ In the North Carolina laboratory and Maryland experiences, the routes of exposure to the toxin(s) elaborated by these *Pfiesteria*-like organisms were through direct contact with the water where a toxic bloom was occurring or through inhalation of aerosols or sprays from these waters.^{3,8} There have been no reports of food-borne illnesses associated with toxic *Pfiesteria* ac-

examined by a dermatologist and skin lesions were biopsied. Pulmonary function tests and laboratory tests, including a complete blood count, blood chemistries, and extensive immunologic studies, were obtained. Each patient was also examined by a neuropsychologist for neurocognitive deficits. Interpretation of tests was by established norms and standards. The principal findings of the medical team included consistent complaints of memory problems. Other symptoms reported by these patients included acute burning of the skin on direct contact with the water, and respiratory irritation. Findings on examination were largely limited to neurocognitive deficits in short-term memory and difficulties in learning. The team identified a total of 11 persons with these problems who had no identified factors that could account for the findings. When the degree and intensity of exposure were examined, the data suggested a dose-response effect.¹² The deficits detected by neurocognitive testing are not readily discernible on routine mental status examination. Until an office-based measure is developed, definitive diagnosis will require referral to a neuropsychologist for psychometric testing.

tivity. The report of the medical team suggests that there are neurocognitive effects in persons with high levels of exposure to an area of the Pocomoke River that was experiencing toxic activity by *Pfiesteria*-like organisms. The medical team's findings formed a base used by public health experts from eight states (Maryland, Delaware, Virginia, West Virginia, North Carolina, South Carolina, Georgia, and Florida) and several federal agencies meeting at the Centers for Disease Control and Prevention to develop exposure and clinical criteria for a preliminary, working definition of possible adverse consequences of exposure to *P. piscicida* or morphologically related organisms (MROs).¹⁴ The combined environmental and clinical signs and symptoms criteria are shown in Table 4. A new report of persistent learning deficits in rats injected with aquarium water that may have contained one or more *Pfiesteria* toxins is consistent with the findings of the medical team.¹⁵

DHMH will continue to gather information about illnesses thought to be related to *Pfiesteria* or *Pfiesteria*-like organisms. Physicians are asked to report any suspected cases to

TABLE 4. CDC consensus criteria - *Pfiesteria* and morphologically related organisms (MROS)- 1997

Exposure criteria:

Exposure to estuarine water characterized by:

- fish with lesions consistent with *P. piscicida* or MRO toxicity (20% of a sample of at least 50 fish of one species having lesions)

OR

- a fish kill involving fish with lesions consistent with *P. piscicida* or MRO toxicity

OR

- a fish kill without lesions, if *P. piscicida* or MROs are present and there is no alternative reason for the fish kill.

Clinical criteria:

- Memory loss

OR

- Confusion

OR

- Acute burning of skin (on direct contact with water)

OR

- Three or more of the following signs or symptoms:
 - Headache
 - Skin rash
 - Eye irritation
 - Upper respiratory irritation
 - Muscle cramps
 - Gastrointestinal complaints (i.e., nausea, vomiting, diarrhea and/or abdominal cramps)

Both the exposure and clinical criteria must be met.

their local health departments. Citizens are asked to report both human illnesses and incidents of fish lesions, erratic fish behavior, or fish kills to the DNR hotline 1-888-584-3110. Citizens should not attempt to investigate these incidents themselves and should avoid lingering in areas experiencing such problems or in areas under investigation. When a report of an illness is made, the local health department will seek to interview the patient to obtain detailed symptom and exposure information. It is anticipated that several epidemiological studies will be undertaken within the context of Maryland's academic, interstate, and federal partnership that will serve to further delineate the extent and duration of health effects. Further laboratory-based research is underway to identify specific toxins affecting human health and develop environmental

assays and bioassays to detect and quantify toxin exposure.

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Diagnosis of *Pfiesteria*-human illness syndrome

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ABSTRACT: *The first case reports of human illness caused by exposure to *Pfiesteria piscicida* toxin(s) acquired outside of a laboratory are reported. Though *Pfiesteria*, a toxin-forming dinoflagellate, is responsible for killing billions of fish in estuaries in North Carolina, its role in human illness has remained controversial, in part due to lack of identification of the toxin. A recent fish kill in the rivers of the lower Eastern Shore has permitted careful investigation and identification of a distinct clinical syndrome resulting from exposure to the *Pfiesteria* toxin — *Pfiesteria* human illness syndrome (PHIS). Patients have memory losses, cognitive impairments, headaches, skin rashes, abdominal pain, secretory diarrhea, conjunctival irritation, and bronchospasm. Not all patients have all elements of the syndrome.*

A cluster of patients have exhibited signs and symptoms after contact with water, aerosol, or water droplets containing toxins from *Pfiesteria piscicida*. *Pfiesteria* is a dinoflagellate with small, toxic forms called zoospores and large nontoxic amoeba forms. Despite the documented presence of *Pfiesteria* in Maryland's Chesapeake Bay, and the Neuse River and Pamlico Sound in North Carolina, there were no prior reports of an association of *Pfiesteria* in the wild and subsequent human illness.

This report is the first to associate *Pfiesteria* toxin exposure with acute human illness. Discharge summaries of five patients I saw fit this presumption. As more is learned, additional information will be pre-

sented, hopefully with details as to etiology, prevention, and therapy.

Case 1

The patient is a 23-year-old white man who had waterskied 500 yards downstream from the site of a fish kill (Shelltown) that occurred the following week. He had been in the water for approximately one hour, having fallen rather frequently. He had been healthy before exposure. Within three hours he began experiencing a severe pounding headache with reduced recent memory, as well as abrupt onset of nausea and dizziness. Because of these symptoms, he retired early. The following morning he awoke with an increasing headache and approximately 30 unusual skin lesions arrayed in an asymmetric distribution on his lower extremities and groin. The lesions, measuring 1.5 cm to 2 cm in diameter, had circumscribed borders that were slightly pruritic, displaying evidence of follicular eruption without evidence of cellulitis. Routine cultures were negative. No fungi, hyphae, or spores were noted. The lesions were similar to those seen by physicians in a laboratory in New Bern, North Carolina.

Skin biopsy showed a significant eosinophilic infiltration with a nonspecific inflammatory response. No induration, central punctum, raised edges, or skin atrophy were noted. Skin lines were maintained and a few petechiae were seen. The patient was treated with a potent steroid topically; no other medications were administered. The tentative diagnosis of human *pfisteriosis* was made.

The patient continued to have severe headaches three days later, at which time the patient also complained of mild shortness of breath. The patient's brother, who had less water exposure on the same day, also noted similar skin lesions. Over the next several days the patient deteriorated, developing a severe stiff neck and changes in his mental status including difficulty in recent memory, mild dysarthria, mild ataxia, and mild difficulty in rapid alternating movements.

The patient was admitted to the local hospital. Computerized axial tomography scan of the brain was normal. Magnetic resonance imaging revealed some mild inflammation of the mastoid and a possible polyp in the sinus. The spinal fluid was clear with a total protein value of 27 and a glucose value of 59, and a simultaneous blood sugar value of 90 (all normal values). Two cells were found. A comprehensive battery of studies done on the spinal fluid did not

show any additional abnormalities. He was treated with nonsteroidal antiinflammatory drugs for his headache; however, his mental status did not resolve. Because of this persistent abnormality, the patient was referred to Dr. Donald Schmechel at Duke University, who has evaluated patients with possible *Pfiesteria*-related illnesses in the past. Dr. Schmechel's evaluation showed persistent defect in memory with specific abnormalities in psychometric testing. He thought the neurocognitive defect was, to a reasonable degree of medical certainty, related to the *Pfiesteria* exposure.

Case 2

The patient is a 26-year-old white man who was swimming in the Pocomoke River on the same day as the waterskier in Case 1. He was somewhat upstream from the Shelltown area. He had an abrupt onset of two lesions, one on his forehead and one on the nose, each approximately 1.5 cm x 2.5 cm. There was a difference in the forehead lesion in this case when compared with the index case. At first glance, this lesion looked like a tinea-type infection but there was no scale, KOH preparation was negative, and the edges were not indurated. The lesion on the nose was more typical of those seen on fish affected by *Pfiesteria*. Biopsy of this lesion showed an eosinophilic infiltrate on a nonspecific inflammatory background. He was treated with topical steroids and made an excellent recovery. There were no additional symptoms or neurologic, respiratory, or gastrointestinal problems. The patient was seen for follow-up on August 13, 1997, with full recovery.

Case 3

This patient is a 30-year-old white woman who was swimming with her children in the Pocomoke River in the vicinity of the waterskier (Case 1). She related that she experienced an abrupt onset of nausea, a profuse watery diarrhea, and headaches after the river exposure. She had a mild loss of appetite. At first, the diarrhea was osmotic (i.e., it was worse after she ate something), but later it became secretory with nocturnal bowel movements. The diarrhea was watery with associated urgency and without blood or mucus. Stool cultures were attempted, but specimens were not satisfactory. Examinations for ova and parasites were negative.

The patient had a sharp periumbilical crampy abdominal pain in association with her headaches, which she described as daily and bimodal. The pain was not associ-

ated with temporomandibular joint pain nor with any trapezius muscle spasm. She had previously been well with neither abnormal social stresses nor abnormal bowel history. She reported that she had been using full bottles of Kaopectate and Pepto Bismol without relief. The patient was treated with Cipro, which is used to treat travelers' diarrhea. After four days her symptoms did not improve. She was given cholestyramine as a bile salt binder. The patient improved quickly over three days. She was well two weeks later. Her headaches cleared as the diarrhea abated.

Case 4

The patient is a Department of the Environment worker who was sorting fish at Shelltown during the active fish kill the first week of August. She was wearing protective gloves extending to her wrists. Her shirt left her forearms exposed, resulting in significant river water splash on this area. Her forearm felt burned; the sensation persisted despite washing with distilled water. Within six hours she began noticing memory problems as well as a productive cough and wheezing. The memory problems were such that she reported she went to the grocery store three times with a list and on each occasion returned home without completing her list because she could not figure out what she was supposed to purchase.

The next day, a rash appeared on her exposed forearm with small blisters that became confluent. Two days later, desquamation of the skin of the right forearm was noted. Her skin healed in about one week. A biopsy was obtained but was nondiagnostic. Because her mental status abnormalities persisted, she was referred to Dr. Schmechel at Duke University for confirmation of possible *Pfiesteria*-related toxic exposure and toxic dermatitis. Her psychometric tests at Duke were similar to those of the laboratory worker exposed to the *Pfiesteria* toxin and the waterskier (Case 1). Dr. Schmechel concurred with the diagnosis.

Case 5

The patient is a 41-year-old state worker whose job of 20 years included sampling shellfish beds for fecal coliforms. He had no history of health problems related to the river. The sampling required that he use a device attached to a rod that was immersed to obtain water samples. He held the rod in his right hand and pulled the wet sample tube off the rod with his ungloved left hand. Soon after exposure on August 5, 1997, he noted development of three lesions but

only on the fingers of his left hand. These went on to desquamate as in the patient described in Case 4. The desquamation was pronounced at the distal tufts and at the posterior nail folds. He had what he called a "hot spot" in his mid-palm that had persisted for the previous two weeks. This lesion was similar to the index case; it was a discrete macule with maintenance of skin lines but with no evidence of scale or any evidence to suggest an insect bite. He felt that the burning in his hand stemmed from the mid-palm macular lesion. When this area was biopsied, he stated that the lidocaine completely stopped the burning for the first time in two weeks.

The patient stated that his memory was reduced from his previous status, pointing out that he had forgotten to call a co-worker, something he normally would not forget to do. He also described a watery diarrhea without blood or mucus which was associated with a crampy periumbilical pain. His skin biopsy was also not diagnostic. He recovered without treatment.

These cases were among others later evaluated by the Department of Health and Mental Hygiene expert physician panel. To date I have referred 35 patients for psychometric testing. The psychometrics give a consistent "fingerprint" of abnormalities seen with Pfiesteria exposure but not with controls. The clinical syndrome is variable but memory loss, secretory diarrhea, conjunctivitis, skin rash, headache, and bronchospasm all may occur.

Continued use of cholestyramine has not only ameliorated diarrhea, but also has helped improve memory loss and asthma-like symptoms.

The lack of illness following eating seafood harvested from the affected waters suggests instability of the toxins in food or destruction of toxins by digestive mechanisms. ■

CASE RECORDS

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A 74-year-old man with persistent fevers

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[USA]

ABSTRACT

An elderly man with a history of extensive world travel presents with a chronic illness and fevers. The febrile illness has been present for eight years, and no diagnosis has been made despite extensive evaluation and testing. The differential diagnosis of this unusual case of fever of unknown origin is discussed.

PRESENTATION OF CASE

A 74-year-old African-American man was admitted after an episode of syncope. He had had several coronary angioplasties performed previously, but had no angina associated with this episode. His electrocardiogram revealed no significant interval change from prior tracings, and an echocardiogram showed normal left ventricular function without segmental wall motion abnormalities. The event was witnessed and no obvious seizure activity was noted. In the emergency room the patient was found to have significant orthostatic hypotension, which responded to intravenous volume loading. The patient gave a history of decreased oral intake and fever. He was admitted for further evaluation.

The patient described an eight-year history of recurrent fevers, with temperature peaks as high as 105°F. The fevers occurred daily between 2:00 and 3:00 p.m., occasionally with chills, shortness of breath, a dry cough, diffuse headache, fatigue, and malaise. Relief was obtained with the use of nonsteroidal antiinflam-

matory agents or acetaminophen. He denied substantial weight loss over this period, and could recall no exposure to tuberculosis. The patient had an extensive travel history that included multiple trips to Africa, the Middle East, and Southern Europe. He denied any significant illnesses during or immediately after these trips and none of his family members, who had accompanied him on these trips, were ill.

The patient had benign prostatic hypertrophy, for which he had undergone a transurethral resection. He had a history of a herniated nucleus pulposus in the lumbar spine. His only medications were isosorbide dinitrate and enteric coated aspirin. He had a distant history of tobacco use and did not drink alcohol. He was retired from military service. He was married and was monogamous.

His temperature was 99.4°F, pulse was 80 and regular, respirations were 20, and blood pressure was 115/50 mm Hg. He appeared comfortable and was thin, but not wasted.

There was no rash or adenopathy, and there were no joint abnormalities. The liver was normal in size and the spleen could not be palpated. A grade I systolic ejection-quality murmur was present along the left lower sternal border. Inspiratory rales were present at the base of the left lung. The remainder of the examination was normal.

Routine serum chemistries, transaminases, and bilirubin, alkaline phosphatase, total protein and albumin, and coagulation parameters were all normal. A urinalysis was normal except for 1+ proteinuria. A 24-hour collection of urine yielded 0.53 of total protein. Hematologic values from admission are shown in **Table 1**.

Routine cultures of blood, sputum, and urine obtained during fever were negative. A 5-tuberculin unit PPD was negative at 48 and 72 hours. A chest x-ray showed blunting of both costophrenic angles (**Figure 1**), and a computed tomogram of the chest confirmed bilateral pleural effusions and showed a mild, nonspecific parenchymal density at the left base without evidence of adenopathy or a mass (**Figure 2**). A gallium scan localized activity to the left lower lobe and pleural effusion. A left thoracentesis was performed and yielded cloudy yellow fluid with a glucose of 82 mg/dl, a total protein of 4.4 g/dl, and an LDH of 610 U/liter. There were 9,225 white blood cells/ μ L (16% polynuclear, 50% lymphs, 24% monocytes, 10% mesothelial) and 6,500 red blood cells/ μ L. Gram's stain and acid-fast stain of the fluid revealed no organisms. Routine culture, as well as culture for acid-fast organisms and fungi, were negative. Cytological examination of the fluid showed no evidence of malignancy. A thoracoscopic biopsy of the left upper lobe, the superior segment of the left lower lobe, and the pleura showed only mild chronic inflammation of the pleura with focal mesothelial hyperplasia.

Thick and thin smears of blood for malaria, and serology for cryptococcal antigens, tularemia, chlamydia, legionella, mycoplasma, and human immunodeficiency virus (HIV) were all negative. IgG antibodies to toxoplasmosis were present. Antibodies to histoplasma, coccidiomyces, blastomyces, and aspergillus were all negative in serum. Rheu-

matoid factor and antinuclear antibodies were not detected in serum. Smith antibodies and antibodies to ribonucleoprotein, SS-A and SS-B (antinuclear antibodies associated with speckled pattern) were not detected in serum.

Fevers continued and the patient was empirically treated with ticarcillin/clavulanic acid and clarithromycin. Fever subsided and the patient felt better. He was discharged on oral ciprofloxacin and clarithromycin. The patient continued to improve at home, remained afebrile, and had increased strength and appetite. He complained of intermittent low back pain and anterior left thigh pain, and a magnetic resonance image of the lumbosacral spine showed a nonspecific wedge-shaped area of low signal intensity on T1-weighted images at L3, with diffusely increased signal intensity of the body of L3 on T2-weighted images. Degenerative disc disease was noted at the L4-L5 level. A three-phase bone scan was performed that revealed mild to moderate tracer accumulation within the vertebral body of L3, which was felt to be atypical for degenerative disease, and the possibility of a metastatic focus raised. Focal soft tissue accumulation in the anterior left thigh was thought to represent heterotopic calcification.

Antibiotics were discontinued after one month. Approximately one week later, daily fevers of greater than 102°F resumed as before, accompanied by a dry cough, rigors, fatigue, headache, anorexia, and a seven-pound weight loss. He was again admitted to the hospital. A detailed history revealed that during the patient's travels dietary precautions were minimal and he described drinking unfiltered water and eating local dairy products, particularly cheeses. He kept no pets and had no significant exposure to farm animals. The physical examination was unchanged with the exception that the prostate was tender, although not en-

TABLE 1. Hematologic values

	<u>Admission #1</u>	<u>Admission #2</u>
White blood cells	12.9 K/ μ L	12.1 K/ μ L
Granulocytes	74.9%	64.4%
Lymphocytes	20.1%	28.4 %
Monocytes	5.0 %	7.2%
Hemoglobin	8.7 g/dl	7.3 g/dl
Hematocrit	25.3%	21.3%
MCV	88.1 fL	86.1 fL
MCH	30.3 pg	29.6 pg
Platelets	259 K/ μ L	154 K/ μ L
Erythrocyte sedimentation rate	—	80 mm/hr.



Figure 1. PA and lateral chest X-Ray, demonstrating bilateral small pleural effusions.

larged, and a possible nodule was palpated in the right lobe.

A computed tomogram of the abdomen and pelvis revealed no evidence of tumor, abscess, or adenopathy. Multiple calcified gallstones were noted, without radiographic evidence of cholecystitis. A transrectal ultrasound of the prostate revealed no evidence of abscess, but showed calcifications within the right lobe. Biopsies of the right and left lobe of the prostate revealed no significant pathology, and the prostate specific antigen was 0.5 ng/ml. A posteroanterior and lateral chest x-ray again showed blunting of the left costophrenic angle and air-space disease at the left base. A fiberoptic bronchoscopy was performed that showed "yellowish plaque-like areas" involving the mucosa of the right middle lobe.

Bronchoalveolar lavage was performed in this area, and the fluid was negative for malignancy on cytological evaluation, and showed no organisms on silver methenamine staining. Routine cultures, and acid fast bacillus and fungal cultures of the lavage fluid were negative. A transbronchial biopsy was

performed in the posterolateral segment of the left lower lobe and showed mild interstitial fibrosis and intraalveolar fibrin deposition. No viral changes were seen on H&E stain, methenamine silver staining for *Pneumocystis* was negative and periodic-acid Schiff-base staining for fungi was also negative.

DISCUSSION

The patient whose case is presented to us today has been vexed by an illness-causing fever for several years. Despite several attempts at diagnosis and therapy, the cause of his illness is unknown. The approach to this problem was first codified in a classical article by Petersdorf and Beeson¹ in *Medicine* written in 1961. These authors created the modern definition of the fever of unknown origin: an illness of more than three weeks duration, with documented fever greater than 101°F, and with no diagnosis after one week of inpatient evaluation. These criteria have been modified by more recent writers, but the original definition serves our purpose well to this day.

As we were taught in medical school, all diagnosis begins with a complete history and physical examination. This is never more true than when approaching a difficult case such as this one. There are three general strategies of diagnostic reasoning techniques used by clinicians: probabilistic reasoning, causal reasoning, and deterministic reasoning.² Causal



Figure 2. CT scan of the chest demonstrating bilateral pleural effusions and a non-specific parenchymal density at the left base.

reasoning, the linkage of cause to effect, and deterministic reasoning, the use of compiled strategies in the form of well-defined rules, will not serve us well in cases such as the one presented to us, in which the

facts of the case are well outside our usual experience. In this case, I will use probabilistic reasoning, the methodical examination of the likelihood of causality.

Categorizing and examining the causes of fevers of unknown origin (FUO) may enable us to grapple with this daunting task. Many authors have periodically reviewed the causes of prolonged fevers that fulfill the criteria of FUO, and the predominance of causes of FUO has shifted over the years. As noninvasive and invasive diagnostic techniques have improved, infectious and neoplastic causes of FUO have diminished in frequency, and those due to other causes have increased in frequency (Table 2).^{1,3}

The single most striking feature of this patient's history is the extreme duration of his febrile symptoms. An FUO of this extreme duration is very uncommon, as untreated infection or neoplasm usually becomes manifest over a prolonged period without appropriate diagnosis or therapy. The fact that this patient has suffered fevers for eight years paradoxically simplifies our task. The first task of the clinician in such a case is to document that fever is actually observed, and if possible, to rule out factitious fevers. In this case, fevers were documented in the hospital. In addition, the findings of pleural effusion, leukocytosis, and anemia all suggest the existence of a truly pathological condition, and militate against factitious fever. Given these facts, a relatively small set of unusual diseases must be considered as causes of FUO of extreme duration⁴ (Table 3). Let us examine some other facts of the case to focus our search further.

The patient is 74 years old; symptoms began at the age of 66. This allows us to eliminate several possibilities. Fevers due to familial Mediterranean fever usually begin in childhood and may be intermittent. Since there is no history of fever prior to age 66 this can be

TABLE 2. Causes of FUO: 1960-1990

■ Infectious	30% to 40%
■ Autoimmune or Hypersensitivity ...	10% to 20%
■ Neoplasm	20% to 30%
■ Miscellaneous	15% to 20%
■ Undiagnosed	5% to 15%

discounted. Similarly, this would be a very advanced age of onset for adult Still's disease, and associated symptoms such as rash, lymphadenopathy, or splenomegaly were not reported. Other collagen-vascular or autoimmune diseases (Table 4) could cause prolonged fever, but again the late onset of disease and the lack of serologic or other symptomatic evidence makes this diagnosis less likely. Polymyalgia rheumatica bears consideration, as it occurs in old age, but none of its classical characteristics — shoulder or hip pain, evidence of inflammatory arthritis, or rapid response to antiinflammatory agents — are reported. Fever induced by medication ("drug fever") is always a possibility, and a trial of discontinuation of medications should always be considered. In this case the patient's medications seemed unlikely to be the cause of the FUO.

Granulomatous hepatitis merits some consideration, as this disease most often develops later in life. Granulomatous hepatitis results from mycobacterial or parasitic infection, or the use of medications such as dilantin, penicillin, or isoniazid, and commonly causes liver function test abnormalities, although cases have rarely been reported in the absence of these findings. Liver biopsy is diagnostic, but my clinical suspicion of granulomatous hepatitis in this case is not high enough to warrant this invasive test.

As the incidence of neoplasm rises in old age, let us consider possible neoplastic causes of FUO. The most common neoplastic causes of FUO are lymphoma, adenocarcinoma, hypernephroma, hepatoma, and atrial myxoma. The duration of this patient's symptoms makes lymphoma or adenocarcinoma unlikely.

TABLE 3. Prolonged FUO (> 1 year)

■ No documented fever	27%
■ Undiagnosed	19%
■ Miscellaneous	13%
■ Factitious	9%
■ Granulomatous hepatitis	8%
■ Neoplasm	7%
■ Adult Still's disease	6%
■ Infections	5%
■ Collagen-vascular disease	4%
■ Familial Mediterranean fever	2%

Computerized tomography of the chest showed only a pleural effusion, and subsequent thoracentesis and bronchoscopy were unrevealing. Imaging of the abdomen and pelvis were performed and, similarly, did not yield a specific diagnosis. A bone marrow biopsy should be done if it was not performed during the recent previous evaluations. All of these

studies should be carefully reviewed to ensure that they were of optimal quality, and if not, they should be repeated. A liver or renal tumor should be detected by such studies. A small atrial myxoma might be difficult to detect, and transthoracic echocardiography should be considered if this has not already been performed.

So we return to consider possible infectious causes of FUO. Several points of this case lead us to be suspicious of infectious FUO at the outset. The extremity of the fevers reported (105°F) and the apparent response to ciprofloxacin therapy lead us to consider an infectious disease. Although a soft sign, the mildly elevated white blood cell count suggests an infectious etiology.

The patient's history includes a striking amount of travel in the developing world and exposure to unfiltered water and unpasteurized dairy products. One can therefore assume that this patient could have acquired virtually any of the infectious pathogens associated with FUO (Table 5). Given the comprehensive evaluation that the patient has undergone, most of these pathogens should have been detected if they were present. However, one pathogen that

TABLE 4. Autoimmune causes of FUO

- Erythema multiforme
- Still's disease
- Polymyalgia rheumatica
- Drug fever
- Hypersensitivity angiitis
- Systemic lupus erythematosus
- Periarteritis nodosa
- Other vasculitis
- Rheumatic fever
- Serum sickness
- Mixed connective tissue disease

is both well known to be difficult to detect and reported to be capable of causing disease for years following its acquisition is brucellosis.

This patient is likely to have brucellosis, a disease caused by a Gram-negative coccobacillus. *Brucella* are divided into several subspecies: *abortus* (cattle), *melitensis* (goats and sheep), *suis* (swine), and *canis* (dogs).

The organism has a worldwide distribution, but is especially prevalent in the Mediterranean (where our patient has spent some time), India, and South and Central America.

B. abortus and *B. suis* are transmitted after contact with livestock; *B. melitensis* is primarily food-borne and transmitted through consumption of unpasteurized dairy products. The slow growth of these organisms in laboratory can hinder diagnosis. Brucellae can require up to four weeks to grow, and the microbiology laboratory will usually have to be asked to hold the sample for prolonged culture.

Brucellosis can involve any organ, but usually localizes to the reticuloendothelial system, typically the hepatobiliary system. It can also involve the bones and joints as a cause of chronic osteomyelitis, the pulmonary system, genitourinary tract, central nervous system, and the cardiovascular system, especially as a cause of chronic endocarditis.

Known classically as undulant fever, Malta fever, or Mediterranean remittent fever, brucellosis was first described in 1859. *B. melitensis* was first isolated from the spleens of British soldiers dying of Malta Fever by Sir David Bruce in 1886. *B. abortus* was first isolated from cattle in 1897, and from a human in Baltimore in 1922. *Brucella* can cause a subclinical, acute, relapsing, or chronic infection. The chronic form of *Brucella* infection can present as an insidious disease, an acute illness followed by relapses (as I believe it is in this case), or a localized infection. Constitutional symptoms are common, with headache, lassitude, depression, insomnia, and other neuropsychiatric symptoms frequently seen. Local-

TABLE 5. Infectious causes of FUO

- | | | |
|---------------------|---------------------------------------|-------------------|
| ■ Bacterial abscess | ■ Cytomegalovirus, | ■ <i>Brucella</i> |
| ■ Salmonella | Epstein-Barr virus, HIV | ■ Osteomyelitis |
| ■ Leishmaniasis | ■ Subacute endocarditis | ■ Malaria |
| ■ Rat bite fever | ■ Toxoplasmosis | ■ Tuberculosis |
| ■ <i>Bartonella</i> | ■ Relapsing fever (<i>Borrelia</i>) | ■ Q fever |
| ■ Lyme disease | ■ Actinomyces | ■ <i>Listeria</i> |
| ■ Histoplasmosis | | |

ized disease may cause a wide variety of symptoms related to the anatomic area involved (for example, osteoarticular symptoms or heart valve dysfunction).

Definitive diagnosis is made by culture or serum agglutination test for specific antibodies. Titers are diagnostic if $\geq 1:160$, or if a fourfold rise is seen on serial samples. Blood cultures are positive in as few as 10% in cases where the infection is old or partially treated, or as many as 85% of acute cases. The frequency of pathogen isolation decreases with duration of infection. Therefore, chronic brucellosis is usually a serologic diagnosis.

In summary, it is most likely that this patient acquired *B. melitensis* by a food-borne route. Exposure to unpasteurized goat, sheep, or even camel dairy products is the most common source of brucella in those who travel.⁵ It is likely that this diagnosis was made by serology. Given the long duration of this infection, I would favor treatment with two drugs rather than one. Although ciprofloxacin has activity against brucellae, fluoroquinolones are not the treatment of choice for this infection, and relapses have been reported following quinolone monotherapy.⁶ Treatment with six weeks of doxycycline and two weeks of gentamycin, or six weeks of doxycycline and rifampin is indicated.

DIAGNOSIS AND CLINICAL COURSE

There was no serologic evidence of Q fever or Lyme disease. *Brucella* antibodies were posi-

tive in a titer of 1:160, which was considered presumptive evidence of current *Brucella* infection. The patient enjoyed cheeses greatly, and on his travels had eaten "fresh" (unpasteurized) cheese and goat cheese, and this was considered to be the probable source of *Brucella* infection. The patient was treated for two months with doxycycline, 100 mg bid, and rifampin, 600 mg/day. The patient's fevers resolved within two weeks of initiation of therapy and exercise capacity improved considerably. After one month of therapy the patient had gained weight and had no fevers greater than 99°F, but described shortness of breath that occurred at approximately 3:00 p.m. daily. After two months of therapy these symptoms resolved, the patient continued to gain weight and had no further fevers. A repeat *Brucella* titer was $< 1:20$.

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Ankle sprains: evaluation, treatment, rehabilitation

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ABSTRACT: Ankle sprains are a common, costly, and potentially disabling problem. The proper history and physical examination will determine the need for radiological evaluation and treatment. Complications of ankle trauma like osteochondral fractures, peroneal tendon injuries, fracture of the os trigonum, synovial impingement, tarsal tunnel syndrome, Achilles tendon inflammation or rupture, and nerve injury are reviewed.

The treatment of ankle sprains is based on the severity of the injury. Treatment begins with rest, ice, compression, and elevation. Casting and orthotics may be needed to facilitate healing. Primary rehabilitation, functional rehabilitation, and performance testing and the assessment of efficacy for each of these modalities are critical parts of proper treatment for ankle sprains.

Introduction

Ankle sprains are a common problem in emergency rooms and primary care practices and, if left unattended, can lead to chronic instability and impairment. Ankle sprains comprise 12% of all injuries seen in emergency rooms and 15% of all sports injuries.¹ Most acute ankle injuries occur in people 21 to 30 years old; however, injuries in the younger and older age groups tend to be more serious.² Forty-four percent of patients have persistent symptoms up to one year after acute, lateral ankle sprains.³

The ankle is a hinge joint that allows approximately 20 degrees of dorsiflexion, 50 degrees of plantar flexion, 10 degrees of adduction-abduction, and 17 degrees of internal-external rotation.⁴ It sustains the greatest load per surface area of any joint in the body.⁵ During heel strike, five times the body weight is placed across the talocrural joint.⁶ The ankle mortise is formed by the articulation of the distal tibia, fibula, and dome of

TABLE 1. History questions

- Time since injury
- Mechanism of injury
- Site of initial pain and swelling
- Skin integrity
- Ability to bear weight immediately and later
- History of previous injury and treatment
- Medical illness affecting neurologic/muscular function

the talus. In neutral position or dorsiflexion the ankle is stable because the widest part of the talus is in the mortise; however, in plantar flexion, ankle stability is decreased as the narrow posterior portion of the talus is in the mortise. During loading of the ankle, articular contact provides 100% of inversion stability and 30% of rotational stability.⁷

The lateral, medial, and interosseous ligaments also contribute to ankle stability and are the elements most frequently injured. Eighty-five percent of ankle injuries involve the lateral ligaments, 5% involve the medial (deltoid) ligaments, and 10% involve the syndesmosis, which comprises the anterior and posterior tibiofibular ligaments and the interosseous ligament.⁸ The three lateral ligaments most commonly injured are the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL). The ATFL is less elastic than the other ligaments and is stretched tighter than the other ligaments in plantar flexion; therefore, an inversion injury with the foot in some degree of plantar flexion most often involves this ligament.⁹ As the foot moves from plantar flexion to dorsiflexion the CFL tightens, so as the severity of the inversion injury or the strength of the forces increases, the likelihood of a CFL injury in addition to an ATFL injury increases. Sixty-six percent of sprains involve the ATFL alone and 20% involve both the ATFL and CFL.¹⁰ Because the PTFL is the strongest of the lateral ligaments, isolated injury to the PTFL is unusual but is often associated with posterior malleolar fractures, which can contribute significantly to a tendency toward internal rotation and posterior instability when the ankle is loaded.¹¹ Eversion injuries of the ankle involve the deltoid ligaments and account for 5% of ankle sprains.¹⁰ This most often occurs as the ankle is forced into eversion or external rotation; for example, when a lateral force is applied to the cleated foot. An eversion or external rotational force sufficient to tear the deltoid ligament completely usually ruptures the syndesmosis or causes a fibular fracture as well.

History

A directed history is useful in identifying ligaments at risk and screening for fractures and other complications. Ques-

tions useful in telephone triage include the time since and circumstance surrounding the injury, the site of initial pain and swelling, loss of skin integrity, whether a pop or snap was heard, ability to bear weight initially and subsequently, and initial treatment (Table 1). While it is optimal to evaluate the ankle as soon as possible after injury, before generalization of swelling and ecchymosis occur, this may not always be possible. Injuries severe enough to cause loss of skin integrity or inability to bear weight warrant immediate evaluation because fractures may be present. Determining the mechanism of injury is of utmost importance. Inversion and eversion injuries were discussed previously. Forced dorsiflexion can result in a sprain of the syndesmosis with pain that greatly exceeds the amount of swelling and that increases with external rotation of the foot. These patients describe pain when the lower leg is squeezed. Forced plantar flexion can result in anterior capsular sprains with pain that worsens on passive plantar flexion and resisted dorsiflexion. Like syndesmosis sprains, anterior capsular sprains can be very slow to heal. A history of injury and subsequent treatments and of the patient's occupational history, activity level, and participation in sports helps to guide treatment and rehabilitation.

Assessment of risk factors associated with ankle sprains can be helpful in determining if a recurrence is likely and possibly preventable. Risk factors include large body mass, impaired sensation or proprioception, history of previous ankle sprains, and muscle strength imbalance. Patients with a history of ankle sprains are twice as likely to have recurrent sprains, particularly if rehabilitation has been incomplete. In a prospective study of college athletes to determine ankle injury risk factors, Baumhauer found increased eversion to inversion strength ratios were greater in injured than in uninjured athletes. In addition, plantar flexion to dorsiflexion strength ratios were greater in the injured leg compared to the uninjured leg of the injured athletes.¹²

Physical examination

Examination of both the injured and uninjured ankles is necessary to detect subtle asymmetries. Examination begins with inspection for swelling and ecchymosis and palpation of sites at risk for fracture or tendon injury, including the malleoli, fifth metatarsal head, navicular, peroneal tendons behind the lateral malleoli, Achilles and anterior tibialis tendons. In addition, the proximal third of the fibula should be palpated in external rotation injuries, which are associated with Maisonneuve fractures, fractures of the proximal fibula. The ATFL can be palpated two to three fingerbreadths anteroinferior to the lateral malleolus, the CFL one to two fingerbreadths inferior to the lateral malleolus, and the PRFL posteroinferior to the posterior edge of the lateral malleolus.

Assessment of range of motion and muscle strength may be limited due to pain in acute injury; however, assessment of neurovascular integrity, gait, and weight-bearing ability is critical for subsequent decision making. A study of physical examination in acute ankle injuries showed the highest interobserver agreement in the patients' ability to bear weight (0.83), fifth metatarsal tenderness (0.78), and malleoli tenderness. Tissue tenderness, swelling, and ecchymosis had the lowest interobserver agreement.¹³

Tests for lateral ligament laxity include the anterior drawer test and the talar tilt test. To perform the anterior drawer test the distal third of the lower leg is held stationary with the nondominant hand while the dominant hand is cupped around the heel. With the foot in neutral position, a force is applied from posterior to anterior at the heel, while feeling for laxity or increased movement within the mortise, comparing the injured side to the normal side. The test is positive in ATFL tears and anterior capsule tears. Peroneal nerve block has been shown to increase the test's sensitivity, but the anterior drawer sign alone is not reliable to detect the full extent of ligament damage.¹⁴ The talar tilt test is performed with the foot in 10 to 20 degrees of plantar flexion. The hindfoot is grasped and slowly inverted as an attempt is made to palpate the lateral aspect of the talus below the lateral malleolus. If the talus is distinctly felt, tilting is occurring at the tibiotalar joint and may indicate a tear of both the ATFL and CFL. The talar tilt test is useful in detecting gross talar instability but because there is a large range of normals, the injured side should be compared to the uninjured side.¹⁵

The clunk test and the squeeze test assess syndesmosis injuries. In the clunk test the hindfoot is moved medially and laterally, avoiding inversion and eversion, while the distal leg is held stationary by the other hand. A clunk can be felt as the talus hits the tibia and fibula if there has been significant mortise widening as in tibiofibular ligament injuries. In the squeeze test, the upper to middle third of the leg is squeezed and pain is felt in the distal third of the leg if the syndesmosis has been compromised. False-positive squeeze tests may be caused by tibia and fibula fractures, calf contusion, or compartment syndrome.¹⁶

A grading system for ankle ligament injuries has been developed and is based on the degree of each injury to each ligament (Table 2). It is limited in that it does not account for grading of injuries to more than one ligament. A grade I sprain

TABLE 2.

Grade	Pathology	Pain and Weightbearing	Stress Examination
I	ligament stretch	minimal pain able to bear weight	normal
II	partial ligament tear	moderate pain weightbearing difficult	anterior drawer and talar tilt may be +/-
III	complete ligament tear	severe pain unable to bear weight	anterior drawer and talar tilt test positive

indicates stretching, without tear, of the ligament. It is accompanied by minimal pain and gait is undisturbed. A grade II sprain is caused by a partial tear of the ligament, is moderately painful and usually accompanied by an antalgic gait. A grade III sprain is caused by a complete tear of the ligament, is severely painful, and may prevent weightbearing.

Radiological evaluation

Before 1990, x-rays were obtained with most ankle sprains. In the last three years, the Ottawa ankle rules have proven to be clinically useful in determining the need for radiographs in acute injury. Patients who are unable to bear weight on injury and during subsequent evaluation or who have tenderness on the posterior edge of the distal 6 cm of either malleolus should have ankle films to rule out fracture. Similarly, patients who are unable to bear weight or who have tenderness of the navicular or the base of the fifth metatarsal should have foot films. These rules have been found to be 100% sensitive and 50% and 77% specific for ankle and foot fractures, respectively. Their use in both hospital and community settings have decreased the use of X-rays by 25% with decreased waiting time, no decrease in patient satisfaction, and a savings of \$600,000 to \$3,000,000 per 100,000 patients.¹⁷⁻¹⁹ The Ottawa ankle rules should not be applied to patients who are pregnant, whose injuries occurred more than 10 days before presentation, or who have isolated skin injuries. Patients younger than 18 years of age are also excluded because open epiphyseal plates predispose to Salter-type fractures.

Ankle plain films should include anteroposterior, lateral, and mortise views, (Figure 1) which are taken in 20 degrees of internal rotation to show the entire talar dome. On anteroposterior and mortise views, the clear space between the edge of the talus and the inner edge of either malleolus should be no greater than the distance between the talar dome and the tibial plafond and no greater than 5 mm. A medial space greater than 5 mm may indicate a deltoid ligament injury, whereas a

lateral clear space greater than 5 mm may indicate a syndesmosis tear.

Ankle stress films are more sensitive in assessing the degree of ligamentous injury. The anterior drawer test is performed as lateral views are taken bilaterally. Five to ten millimeters of talar movement usually indicates an ATFL tear, whereas 11 to 15 mm may indicate ATFL and CFL tear and more than 15 mm may indicate ATFL, CFL, and PTFL tears. The talar tilt test is performed as mortise views are taken bilaterally. Approximately 10 to 20 degrees of tilt may be seen with ATFL tears, whereas more than 20 degrees of tilt may indicate a combined ATFL and CFL injury. Because there is a great degree of normal variation in the degree of talar tilt (0 to 27 degrees) a side-to-side variation of greater than 10 degrees also signifies a positive test.²⁰

Arthrography, if used within two to three days of acute injury, can accurately detect ligament damage with a sensitivity that approaches 100% and few false positives. If extravasation of the dye occurs in to the syndesmosis, ATFL injury is likely, whereas extravasation near the tip of the lateral malleolus and into the peroneal tendon sheath indicates probable CFL injury. Extravasation of dye at the medial malleolus is consistent with deltoid ligament injury.^{21,22}

Computed tomography (CT), bone scans, and magnetic resonance imaging (MRI) are used less often in acute injury. CT shows the bony elements of the ankle well and bone scans can show occult fractures and synovial pathology. Both may be more useful in evaluation of persistent or chronic ankle pain after acute sprain. MRI gives accurate anatomic information about the ligaments and tendons of the ankle and correlates

well with stress x-rays but does not replace physical examination and stress x-rays in providing information on the degree of instability caused by the sprain.²³ MRI may be especially useful if a double ligament tear is suspected and surgery is being considered or in the diagnosis of osteochondral lesions or talar dome fractures.

Arthroscopy is not useful acutely but may be helpful in the evaluation of chronic ankle pain. It can be used to diagnose talar dome abnormalities, loose bodies, and ossicles within the ligaments.

Complications

Complications of ankle sprains are not uncommon and can cause significant impairment and chronic pain. Osteochondral fractures complicate 6.5% of all ankle sprains and occur when the talus abuts the tibial plafond. It can occur with any type of sprain but should be suspected with marked inversion injuries. It is easily missed on early x-rays and is often diagnosed four to six weeks or up to a year after a sprain that fails to heal.²⁴ Patients experience swelling, a sensation of catching and locking of the joint, difficulty pushing off, and pain that increases with activity and is at the joint line in the upper medial or lateral corner of the joint. Diagnosis can sometimes be made with mortise views; however, CT and MRI are more sensitive. Treatment consists of casting and nonweightbearing for six weeks initially followed by surgery to excise the fragment if symptoms persist.

Peroneal tendon injuries range from tendonitis to rupture. Peroneus brevis and longus tendons track behind the lateral malleolus, held in place by a retinaculum, and act to plantar-



Figure 1. Ankle plain films.

flex and evert the foot. The peroneus brevis inserts at the base of the fifth metatarsal, whereas the peroneus longus inserts on the head of the first metatarsal. Sudden dorsiflexion after the peronei have contracted can rupture the peroneal retinaculum, causing popping of the tendons over the lateral malleolus on plantar flexion and eversion, or weakness on plantar flexion and eversion if the tendons themselves have ruptured. The patient experiences point tenderness and swelling behind the lateral malleolus and pain on dorsiflexion. Surgery has a role in acute injury but may not be necessary in a chronic injury. External support does not compensate well for this type of injury. Avulsion of the fifth metatarsal head by the peroneus brevis tendon may occur in inversion injuries and is suspected with localized tenderness and swelling. It should not be confused with a Jones fracture, which is a diaphyseal fracture of the fifth metatarsal.

Fractured os trigonum is another cause of chronic ankle pain following acute sprain. Fourteen percent of people have a sesamoid bone posterior to the talus, called the os trigonum. Patients often give a history of a previous ankle sprain with normal x-rays, persistent posterior and lateral ankle pain and swelling, and a sensation of giving way. On examination there is approximately a 25-degree decrease in plantar flexion, tenderness to palpation posterior to the tibia and anterior to the Achilles tendon, and increased pain with forced plantar flexion or resisted plantar flexion of the hallux.²⁵

Synovial impingement may be a cause of persistent anterolateral pain after acute injury. The synovium is pinched at the articulation of the talus with the tibia and fibula. The patient experiences pain and swelling intermittently, usually associated with activity. On examination there is tenderness anteriorly and a feeling of pinching with forced dorsiflexion. Diagnosis is made with MRI or arthroscopy. Anterior capsule sprains are caused by forced plantar flexion (for example, sliding into a base with a plantar-flexed foot). The patient has pain on resisted dorsiflexion and passive plantar flexion and may have a positive anterior drawer test. These injuries are slow to heal.

Tarsal tunnel syndrome is a nerve entrapment of the posterior tibial nerve behind the medial malleolus, which causes a dysesthetic pain over the medial arch and posteromedial aspect of the foot. There may be a positive Tinel's sign at the posterior aspect of the medial talus.

Achilles tendonitis or rupture is a complication of ankle sprains. The distal 6 cm of the tendon is not well vascularized and is the most common site of injury.²⁶ Achilles tendonitis is experienced as local tenderness, crepitus, and pain on passive dorsiflexion and resisted plantar flexion. In Achilles tendon rupture, patients may hear a pop and notice weakness on plantar flexion. If the tendon is completely ruptured, the

foot will not plantar-flex when the calf is squeezed (positive Thompson's test).

Nerve injury can accompany more severe sprains. The deep peroneal nerve innervates the anterior tibialis and toe extensors in the anterior compartment of the lower leg. The superficial peroneal nerve innervates peroneus longus and brevis in the lateral compartment and the tibial nerve innervates gastrocnemius and soleus as well as the toe flexors and posterior tibialis muscles. Electromyography performed two weeks after acute ankle injury demonstrate peroneal nerve injury in 86% and posterior tibial nerve injury in 83% of subjects with grade III sprains (complete tear of at least one ligament).²⁷

Treatment

Initial treatment of the acute ankle sprain is governed by the RICE principles: relative rest, ice, compression, and elevation. Relative rest allows movement and activity that does not cause pain. A prospective study of first ankle sprains comparing immobilization and nonweightbearing with early mobilization and weightbearing as tolerated showed similar reinjury rates but the early mobilization group had less pain and substantially earlier return to work rates.²⁸ Cryotherapy decreases histologic evidence of inflammation by inhibiting histamine, polymorphonucleocyte, and collagenase activity, and causes vasoconstriction, resulting in decreases in temperature of skin and subcutaneous tissues and, to a lesser degree, muscle. Decrease in muscle and intraarticular temperature is maintained for several hours after removal of the cooling agent. Prolonged application of cold, however, can result in a sympathetically mediated reflex vasodilation in an attempt to rewarm the area, which may actually worsen swelling.²⁹ Current recommendations are to apply ice packs for 20 minutes every two hours. Cold packs kept in a deep freezer take on the temperature of the freezer in which they are stored and should be used in shorter sessions. Cryotherapy is contraindicated in Raynaud's phenomenon and cold-induced allergy, and application over superficial nerves should be avoided. Compressive dressings are most useful in the first 48 to 72 hours and should be reapplied every four hours being careful not to cause obstruction to distal venous return.

Grade I sprains can be adequately treated using the above principles followed by a home exercise program. Return to sports can be achieved in one to two weeks if bracing is considered for high-risk sports such as basketball and soccer. Complete healing should be expected in four to six weeks. Patients with grade II sprains should be placed on crutches until they are able to walk comfortably. The ankle should be immobilized at 90 degrees for two to three weeks using an orthosis or splint. Physical therapy should be initiated after

the acute phase and orthopedic referral considered in athletes. Eight to twelve weeks is required for complete healing. Grade III sprains should be immobilized three to eight weeks with a removable ankle foot orthosis or short walking casts. Eighty to ninety percent of lateral ligament sprains can be treated nonsurgically but orthopedic referral is warranted in these patients. Ten to twenty percent may eventually require delayed repair or reconstruction to regain functional stability.³⁰ Aggressive rehabilitation is needed for six to eight weeks. Immediate orthopedic referral should be obtained in compound, displaced, or malleolar fractures. Orthopedic consultation may be needed if there is a history of recurrent sprains or if a nondisplaced fracture is present. Of note, eversion injuries causing deltoid ligament sprains can be accompanied by syndesmosis sprains and should be x-rayed two days after the original injury, looking for diastasis of the tibia and fibula.

Orthotics or braces are important in both the initial treatment and prevention of ankle injuries. Acutely, their role is to compress, protect, support, and limit range of motion of the injured ankle, most importantly plantar flexion, which is a precarious position of an injured ankle. Major categories include tape, lace-up braces, semirigid orthoses, and plaster posterior splints. Ankle taping has been shown to decrease the incidence of ankle sprains. Initially, it restricts range of motion but loses 40% of its net support in the first ten minutes of vigorous activity and after one hour no longer restricts range of motion.^{31,32} It is believed to improve proprioception because taped ankles have shorter peroneal reaction times, possibly providing earlier dynamic stabilization.³³ Lace-up braces have been shown to be more effective than tape in preventing ankle injuries and can be tightened during activity.³⁴ Semirigid orthoses (e.g., Aircast) come in a variety of styles including those with pneumatic inserts and hinged ankles. The Aircast Sport Stirrup has been shown to decrease the incidence of acute and current ankle sprains and did not significantly affect sports performance.³⁵ In a study comparing the use of compression bandages to semirigid orthoses in acute ankle sprains, the brace group was more mobile in the initial phase of rehabilitation and were able to return to work earlier than the compression bandage group.³⁶

Orthoses are also indicated for prevention of ankle injuries, particularly in people with poor flexibility, weakness, and decreased proprioception, especially when engaging in high-risk activities. A randomized study of soccer players with a history of ankle sprains showed a fivefold decrease in the incidence of recurrent ankle sprains with the use of semirigid orthoses.³⁷ Athletic shoe height can increase the active resistance to an inversion moment when the foot is in plantar flexion and can increase the passive resistance afforded by tape and orthoses.³⁸

People who have had one ankle sprain have a twofold increase in the likelihood of reinjuring the same ankle,³⁹ and it is estimated that 30% to 60% of patients develop chronic mechanical instability.^{40,41} Chronic instability has been shown to result in degenerative arthritis.⁴² A 1 mm displacement of the talus reduces the ankle's weightbearing surface by 42%, which may contribute to these degenerative changes and promote instability.⁴³ Biomechanical studies of patients following ankle sprains have shown various abnormalities including altered hip muscle recruitment patterns, longer reaction times in peroneus longus and tibialis anterior, and earlier activation of tibialis anterior compared to peroneal muscles, a reversal of the normal pattern.⁴⁴⁻⁴⁶ These findings might suggest that ankle sprain patients alter their proprioceptive response to postural perturbations, which also may contribute to instability. The most common cause of reinjury after ankle sprains and chronic instability is incomplete rehabilitation.

The most important factor in prevention of subsequent ankle sprains is complete rehabilitation, which should include exercises to increase peroneal strength and improve proprioception, which contributes to dynamic stabilization and injury prevention.⁴⁷ Slow, static stretching of the calf muscles has been shown to decrease the severity of subsequent ankle injuries.⁴⁸

Surgical treatment

Surgical evaluation should be obtained in lateral ankle sprain patients who have a third-degree sprain with a positive talar tilt sign greater than 15 degrees and an anterior drawer sign greater than 1 cm. A grade III ankle sprain is not an absolute indication for surgical stabilization, but should be evaluated by a surgeon.⁴⁹⁻⁵² Hemarthrosis of the ankle joint is also an indication for surgical referral.

Rehabilitation

Intensive rehabilitation of ankle injuries has three major components: primary rehabilitation, functional rehabilitation, and functional performance testing. The goal of primary rehabilitation is to provide modalities and exercises to improve range of motion, strength, and proprioception. Physical therapists assess both active and passive range of motion, taking into account not only the amount of range, but the end feel of the joint at the extremes of its range of motion and the patient's pain in relation to restricted motion, before initiating therapeutic exercise. Neurophysiologic stretching is useful in stretching tight muscles and tendons. It begins with an isometric contraction of the tight muscle followed by relaxation of that muscle and contraction of the antagonist muscle while the tight muscle is lengthened with a manual stretch. In

addition, patients are often given self-stretch exercises, emphasizing heel cord stretching, with instructions to hold the stretch for 30 to 60 seconds and avoid bouncing to minimize activation of the muscle spindle and stretch reflexes.⁵³

Strengthening exercises are of three types, all of which improve isolated muscle function, a necessary precursor to improving muscle performance in functional patterns. An isometric exercise commonly used to strengthen the peroneals involves pushing the lateral aspect of the forefoot against a fixed surface. Exercises using Therabands, a series of rubber bands of graduated strengths, provide isotonic exercises for strengthening dorsiflexors and evertors. Isokinetic exercise is achieved using Cybex machines.

Proprioceptive exercises are especially important for full functional return and injury prevention. Injury has been shown to result in partial deafferentation with sensory deficits that affect joint position sense and joint movement. These deficits may play a role in chronic injury and reinjury.⁵⁴ Examples of proprioceptive exercises include the modified Romberg test, in which the patient balances on the injured leg with eyes open and then closed for at least one minute. Other examples include the use of a tilt board, and use of ankle discs, also known as a BAPS board.

Functional rehabilitation may begin when the patient is able to walk without pain or gait deviation on flat surfaces and stairs and when range of motion is full and painless. Its purpose is to return to preinjury level of activity while minimizing the risk of reinjury. At this point a transition is made from the use of open-chain strengthening exercises, which isolate movement at a given joint, to closed-chain exercises, which integrate the actions of agonist, antagonist, and synergistic muscles needed for stabilization and ambulation. Squats are an example of a closed-chain exercise for the lower extremities.

The primary goal of functional rehabilitation is the restoration of functional stability, which is the ability to control the translation of the ankle during dynamic functional activity. Except in surgical cases, stability is reestablished by the dynamic muscular component of the ankle, which requires retraining of the ankle's complex neuromotor patterns. This must be restored before a patient can perform sport-specific skills safely and efficiently.⁵⁵ Exercise programs begin to use sport-specific movement patterns to improve functional strength and kinesthesia, proprioception, eccentric loading, and aerobic conditioning and allow for gradual resumption of sports activities.

In phase one of a functional rehabilitation program, a patient would walk or jog on flat surfaces, ascend and descend stairs forward and backward, and bike or swim for conditioning. Phase two would add exercise to allow turning, changing

directions and lateral movements while running, and eccentric loading with stair running. Phase three would add cutting drills, shuttle runs, carioca crossover drills, and running for aerobic conditioning. In phase four, sports-specific activities such as lay-ups, dribbling, and fielding would be added and running continued for conditioning.

Functional performance testing assesses readiness to return to a specific sport using tests to assess the functional stability and dynamic capability of the injured joint. Tests found to correlate well with good recovery are descending stairs, walking on heels and toes, and balancing on a square beam.⁵⁶ Other tests studied in anterior cruciate ligament injuries, but possibly useful in evaluation of the ankle, include the ability to run figure-of-eight patterns, single-leg hop test for one minute, carioca crossovers, and shuttle run.⁵⁷

Semirigid ankle orthoses have been shown to prevent recurrent sprains and protect ligament reconstructions,⁵⁸ however, the need for them can be obfuscated by complete rehabilitation in most patients.

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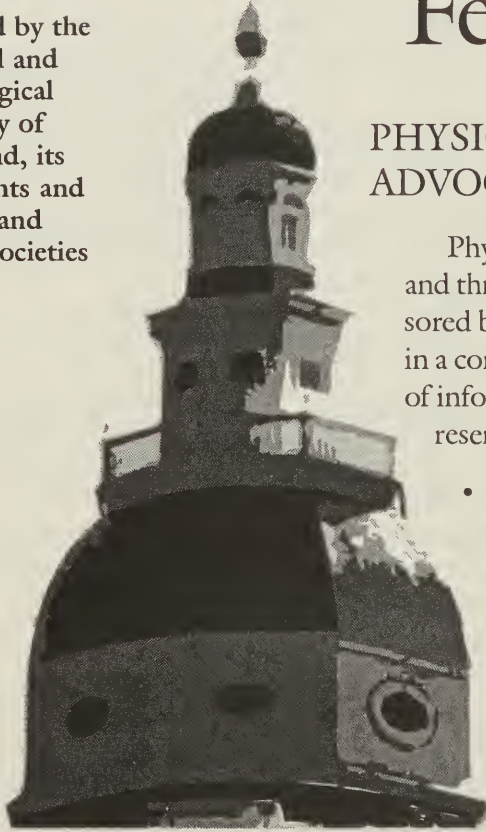
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MARYLAND MEDICAL HISTORY

Heroic medicine, bloodletting, and the sad fate of George Washington

ABSTRACT

Bloodletting was an established medical treatment for more than two millennia, yet its greatest impact in the United States is undoubtedly the role it played in the treatment of President George Washington. The theoretical justification for bloodletting is provided, followed by a detailed description of the treatment Washington received, reflecting the role played by heroic medicine in the American president's demise. The arguments of bloodletting's critics emerge as well founded; indeed, Washington and many others might have suffered less had heroic medicine not been applied.

Most societies practiced bloodletting for about 2,500 years, but few are aware of the central role it played in the treatment of George Washington. America's first president fell ill and was treated—with tragic and fatal consequences—by the prescribed methods of venesection and purging that had long dominated Western medicine. The theoretical justification for these methods derived from the belief that human health was maintained by a balance among four humors within the body: blood, phlegm, black bile (melancholy), and yellow bile (choler). Medical practitioners believed that diseases were caused by an imbalance between or overabundance of these humors, which logically led them to prescribe a course of treatment that encouraged evacuation, whether by bloodletting, inducing vomiting, purging, or diuresis. This type of treatment, which reached its peak during the end of the eighteenth and beginning of the nineteenth centuries, was commonly referred to as “heroic” medicine because of the large amount of strong medications administered to the patient, accompanied by excessive bloodletting.^{1,2} Ironically, Washington's illness occurred shortly before major scientific discoveries forever destroyed the theoretical foundations of heroic medicine.

Heroic medicine originated in the 5th century B.C. during the time of Hippocrates, who recommended venesection for the treatment of various diseases. Bloodletting was subsequently endorsed by other medical authorities of the ancient world, such as Galen (130-200 A.D.), who claimed to be an intellectual heir of Hippocrates. With such authoritative support, bloodletting and heroic medicine became firmly established treatment modalities that survived for another 1,500 years. In colonial America, the practice was supported by several prominent physicians, including Benjamin Rush (a signer of the Declaration of Independence), who went so far as to recommend treating all of his patients with massive bleedings and copious purging.

Despite its historical predominance over other forms of treatment, bloodletting was never without influential and authoritative critics. The Greek physician Erisistratos (300-260 B.C.) took issue with Hippocrates, arguing that bloodletting was dangerous because of the difficulty in estimating the amount of blood to be withdrawn, as well as the possibility of mistakenly cutting other vital structures such as tendons, arteries, nerves, etc. In the new American republic heroic medicine was similarly attacked by physicians, as well as by such medical groups as homeopaths, botanics, and water cure therapists, who claimed their methods caused no harm and patients fared comparatively better under their care. The decisive moment in this debate came with the demonstration by Louis Pasteur (1822-

1895) and Robert Koch (1843-1910) that many illnesses were caused by microorganisms, against which bloodletting was ineffective. The practice of heroic medicine declined rapidly during the latter half of the nineteenth century.

Perhaps most remarkable was that bloodletting was accepted by most medical authorities of the Western world for such a long time, even though it had been categorically rejected by Native American societies, whose views were deemed too "uncivilized" for Western tastes. The *Water Cure Journal* best captures the sentiment of the indigenous Americans toward this type of treatment:

Lafontaine, [sic] in his voyage to North America ... says: "the Indians are yet more astonished at our custom of bleeding, for" say they, "the blood being the tapet of life, we have more occasion to pour it in than to take it out, considering that life sinks when its principle cause is moved off, from whence, 'tis a natural consequence, that after loss of blood, nature acts but feebly and heavily, the entrails are overheated, and all the parts are dried..."³

Indeed, Washington might have been better served if left to the devices of Native Americans or water cure therapists, as reflected in the description of the management of his illness with the methods of heroic medicine. His short, fatal illness in 1799 is described by James Craik, attending physician, and Elisha E. Dick, consulting physician; the account, considered to be of interest to the "general public and professionals" of the time, is even more interesting and revealing now:

Some time in the night of Friday, the 13th inst., having been exposed to a rain on the preceding day, General Washington was attacked with an inflammatory affection of the upper part of the windpipe, called, in the technical language, *Cynanche Trachealis*. The disease commenced with a violent ague, accompanied with some pain in the upper and fore part of the throat, a sense of stricture in the same part, a cough, and a difficult, rather than a painful, deglutition, which was soon succeeded by fever, and a quick and laborious respiration. The necessity of bloodletting suggesting itself to the General, he procured a *bleeder* in the neighborhood, who took from his arm 12 or 14 ounces of blood. He could not, by any means, be prevailed on by the family to send for the attending physician, until the following morning, who arrived at Mount Vernon at about ten o'clock on Saturday. Discovering the case to be quite alarming, and foreseeing the fatal tendency of the disease, two consulting physicians were immediately sent for, who arrived, one at half after three and the other at four o'clock in the afternoon. In the meantime were employed two pretty copious bleedings, a blister was applied to the part affected, two moderate doses of calomel were given, and an injection was administered, which operated on the lower intestines, but all without any perceptible advantage, the respiration becoming still more difficult and distressing. Upon the arrival of the first consulting physician, it was agreed, as there were yet no signs of accumulation in the bronchial vessels of the lungs, to try the result of another bleeding, when about 32 ounces of blood were drawn, without the smallest apparent alleviation of the disease.

Basins of vinegar and water were frequently inhaled, 10 grains of calomel were given, succeeded by repeated doses of emetic tartar, amounting in all to five or six grains, with no other effect than a copious discharge from the bowels. The power of life seemed now manifestly yielding to the force of the disease; blisters were applied to the extremities, together with a cataplasm of bran and vinegar to the throat. Speaking, which was painful from the beginning, now became almost impracticable; respiration grew more and more contracted and imperfect, till, at half after eleven, on Saturday night, retaining the full possession of his intellects, he expired without a struggle.

He was fully impressed at the beginning of his complaint, as well as throughout every succeeding stage of it, that its conclusion would be mortal, submitting to the several exertions made for his recovery, rather as a duty than from any expectation of their efficacy. He considered the operations of death upon his system as coeval with the disease; and several hours before his death, after repeated efforts to be understood, succeeded in expressing a desire that he might be permitted to die without further interposition. During the short period of his illness, he economized his time in the management of such few concerns as required his attention, with the utmost serenity, and anticipated his approaching dissolution with every demonstration of that equanimity for which his whole life has been so uniformly and singularly conspicuous.⁴

This review of the fatal illness of America's first president is of interest in several respects. First, Washington's illness does not resemble pneumonia, as some historians have suspected. Nor was it likely to have been strep throat because of the fact that he lost his voice as the illness progressed—a symptom that indicates the larynx was probably affected. Moreover, strep throat is not likely to develop so rapidly. It appears that the most plausible diagnosis of his illness was bacterial epiglottitis. Second, the treatment Washington received clearly was misdirected and may have contributed to his rapid demise. Even Dr. Craik, who attended to Washington in his final hours, subsequently concluded that his patient might have expired because of the removal of too much blood.² This vignette suggests that George Washington might have had a better chance of survival if the methods of heroic medicine had not been used.

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PETER STAVRAKIS, M.D.

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Interviews with women medical society leaders

In this article some of Maryland's more active female physicians are discussed. They have been instrumental in molding policy, as well as Med Chi's legislative agenda, over the past few years. These women physicians are our professional societies' leaders; they are involved on multiple levels within the component and state organizations. Although these women are vocal and participate on many committees and in the House of Delegates, they sometimes are not visible to the members, as few have been elected officers on the state level.

Several questions were posed to these leaders, who represent various geographical areas of the state and a range of different specialties. Their responses reveal the diversity of our society and the talents that represent organized medicine.

Medical contributions

Maryland's female physician leaders have made some significant contributions to the field of medicine as evidenced by the following achievements. Margaret Mullins, M.D., president of the Anne Arundel County Medical Society during 1995 and 1996, a Legislative Committee member for 13 years (subcommittee chair for one year), and practitioner of internal medicine and acupuncture, was inspired to introduce complementary medicine to physicians and is very active in Med Chi's Complementary Medicine Committee.

One of medicine's leaders in the field of ophthalmology, Gerri Goodman, M.D., from the Baltimore City Medical Society (past chair of the component's Young Physicians Committee and member of the Public Relations Committee), is a past president of the Maryland Ophthalmology Society and has met the challenge of combining a clinical practice with involvement in resident teaching and participation in organized medicine activities. Dr. Goodman was instrumental in the opening and oversight of the Greater Baltimore Medical Center's Excimer Laser Radial Keratectomy Program.

The following two physicians have been very active in tobacco control legislation. Eva Smorzaniuk, M.D.,

past president, vice president, and secretary/treasurer of Talbot County Medical Society, Med Chi delegate, and a practicing diagnostic radiologist at Easton Memorial Hospital, demonstrated vocal and visual presence during the heated discussions of her county's referendum on smoking bans in public establishments. She gave many lectures, addressed radio programs, and felt rewarded for her efforts as the public opinion favored her position. Although promoting public health issues seems a little unusual for a radiologist, Dr. Smorzaniuk's comments provide insight into her actions: "I feel physicians must realign themselves with patients and regain their trust. Part of this requires us to demonstrate we are concerned with public health and well-being," she explains.

Katherine Farrell, M.D., of the Anne Arundel County Health Department, also played an active leadership role in passing the most stringent smoke-free workplace laws in existence. Dr. Farrell is a founding member of the Public Health Committee of the Anne Arundel County Medical Society, a member of Med Chi's Public Health Committee, and a board member of the Smoke Free Maryland Coalition. Her specialty is public health and preventive medicine.

One of our most noticeable women leaders is Carol Garvey, M.D., who has an impressive record of involvement in organized medicine, on both the state and local levels. She has been Montgomery County component society president, president-elect, chairperson of the Public Health Committee, member of the component board, and at Med Chi, she has served as treasurer, secretary, member of the board of trustees, chairperson of the Finance Committee, AMA Alternate Delegate, chairperson of the Ad Hoc Subcommittee to Study Physician Offices, and a member of the Med Chi Agency Board of Directors, among many other positions. She was listed in *Washingtonian Magazine's* 1987 article on "Good Family Doctors: The Pediatricians, Internists, and Family Practitioners that Other Physicians Would See if They Needed Help." One of her most significant accomplishments to date has been the formation of the Primary Care Coalition of Montgomery County. The

coalition has received a Robert Wood Johnson Foundation grant to create a service for low-income patients which refers them to physicians willing to accept reduced fees. Dr. Garvey is now the county health officer for Montgomery County.

Zorayda Lee-Llacer, M.D., a board certified anesthesiologist, is medical director of the medical surgical intensive care unit at Doctor's Hospital of Prince George's County and a member of the Board of Physician Quality Assurance. She has been active in organized medicine as president-elect of the Prince George's County Medical Society, a Med Chi delegate since 1986, and a member of the IPA, Long Term Care and Geriatrics, Library and History, and Reference committees, and is now co-chair of Med Chi's new Committee to Investigate Discrimination Complaints. Dr. Lee-Llacer is also director and president of the National Provider Corporation, an integrated health care management company, and has published a chapter concerning respiratory intensive care management in the *Medical Surgical Nursing Textbook*.

A very active member in the field of environmental and occupational health, Susan Guarnieri, M.D., is manager of safety and medical services at Baltimore Gas & Electric Company, where she has been employed since 1987. Before that, Dr. Guarnieri was Commissioner of Health for Baltimore City from 1984 to 1987. She is past president of the Baltimore City Medical Society and Edison Electric Institute Medical Consultants, past chair of the Governor's Commission on Women's Health, and current delegate to Med Chi. She has served as vice president of the Baltimore City Medical Society Foundation Board of Directors, and is past vice chair and commissioner of the Health Services Cost Review Commission. Dr. Guarnieri also finds time to fill the role of resident preceptor and advisor at The Johns Hopkins School of Public Health, along with participating on the board of directors of the Maryland Occupational Medical Association. Another noteworthy entry on her curriculum vitae is medical news commentator from 1977 to 1979 for WMAR-TV on the show *Prognosis*.

Cheryl Winchell, M.D., a family practitioner in solo practice since 1973, has been active in organized medicine as well as being very involved with the Board of Physician Quality Assurance (BPQA). She was

appointed to the BPQA in 1991 and currently serves as executive secretary/treasurer, editor of the *BPQA newsletter*, and was BPQA's liaison to the Governor's Task Force on Professional-Client Sexual Exploitation. Some of the issues she has dealt with are CDS prescribing, adequate medical record documentation, duties of unlicensed medical assistants, and proposals for a nonpublic, nondisciplinary tract for physician violations such as poor record keeping and minor breaches of a non-willful nature. Dr. Winchell has been the chief of staff at Shady Grove Adventist Hospital, past president and treasurer of the Montgomery County Medical Society, and chair of the component Legislative Committee. She is now a member of the Med Chi Legislative Committee and was cited by Washingtonian Magazine in 1989 as one of the "100 Most Powerful Women in Washington," in 1993 as one of the "970 Top Washington Physicians," and in 1995 as one of the physicians in the article "Top Doctors: Physicians Name the Specialists They'd Go To."

Health care delivery concerns

The first question we posed to our leaders was, "What do you think are the most pressing issues with respect to the current delivery and payment systems for health care in this state?" The responses overwhelmingly centered around the intrusion of insurance and managed care companies into the provision of health care services.

Dr. Farrell, believes, as did most physicians who were interviewed, that "quality has taken a back seat to cost savings and that quality needs to be considered first." Dr. Garvey concurs that quality has been forsaken in the name of cost. "Delivery and payment systems in Maryland and elsewhere are in chaos because insurers seek the least expensive physicians and employers seek the least expensive insurers, causing longstanding doctor-patient relationships to be disrupted. I am convinced that at some point the pendulum will swing away from cost as the primary, if not sole, force shaping medical care, but until then things will continue to be pretty uncomfortable both for doctors and for patients," Dr. Garvey explains.

One of Maryland's most beloved and respected mentors, Margaret Snow, M.D., secretary of the Montgomery County Medical Society, past chair of the Women in Medicine Committee and AIDS Committee, member of Med Chi's Ethics and Legislative committees, and a

practicing family practitioner, also agrees that the most pressing issues facing physicians include excessive government and insurance company intrusion into the practice of medicine. "My patients are shifted from insurance company to insurance company like sheep in a pen. This lack of continuity destroys the unique personal relationship of doctor and patient," says Dr. Snow.

Dr. Marianne Benkert, past president and Executive Committee member of the Baltimore County Medical Association, and Med Chi delegate and chair of the Council on Ethical and Judicial Affairs, explains how managed care has disrupted her practice of medicine. "In psychiatry, we have to receive authorization for our services and submit periodic treatment plans in order to justify continued service. Every aspect of our treatment is subject to scrutiny. It is with sadness that I see the professionalism of physicians being eroded. Although we have the medical expertise, we are being deprived of one of the privileges of the professional — the decisions of how to use that expertise."

Dr. Lee-Llacer believes there is a solution to the havoc managed care has wreaked. It involves educating legislators as to the struggles physicians are experiencing, such as economic credentialing, pre-authorizations, excessive paperwork, and administrative overhead. "Physicians should lobby to make restrictions easier to meet so that physician groups can compete in the market. Eventually direct contracting between business and the providers in a competitive market will cut costs. Physician groups will assume risk by guaranteeing the delivery of care, which encompasses not only physician services but includes hospitalization, DME [durable medical equipment], prescriptions, diagnostic services, et cetera. A physician group that is efficient and vertically integrated has a better chance to survive and get contracts," Dr. Lee-Llacer explains.

Changes in the field of medicine

Our next questions were "What are the most significant changes in medicine (technology, delivery, managed care, etc.) that have occurred throughout your career in medicine? Have these changes had a positive or negative impact on the profession?" Technology and managed care were cited by those surveyed as having the most impact on the landscape of American medicine in recent years.

Dr. Goodman believes the existence of technology has fueled the problems with managed care. "We face many new challenges that have appeared due to the advances we have made in technology and the problems that exist with the current state of the health care delivery system. I feel that there is not enough physician representation and input into the way managed care is developing. There has been a devaluation of the physician by the media and society and an emphasis on economy, quantity, and cost-effectiveness. The HMOs and insurance companies have not made changes that have helped the patients and they are causing stress in the medical community by increasing our paperwork, limiting our access to patients (and vice versa), and controlling decision making in medical care. I feel that we have a big challenge in front of us, trying to work on this issue so that the quality of the U.S. health care system, that we have worked for years to create, is not jeopardized."

Sharon Pusin, M.D., member of the board of the Baltimore County Medical Association (BCMA), chairperson of the Ad Hoc Committee on Managed Care BCMA, Legislative Committee member and delegate to Med Chi, has been a practicing ophthalmologist for 20 years in private practice. She also believes technology has lent significant changes to her field, but more with regard to procedures such as cataract surgery, laser treatment of diabetic retinopathy, and medical and laser advances in glaucoma management. "With posterior chamber intraocular lenses, we can provide safe and seemingly miraculous restoration of vision in cataract patients. We have replaced the thick spectacles which caused image distortion and magnification, leading to falls and hip fractures in the elderly. In patients with diabetes mellitus, the incidence of blindness has been significantly reduced by laser treatment guided by the results of nationally conducted clinical trials. Pharmaceutical research has increased our armamentarium of drugs to treat glaucoma. Being on the cutting edge has made ophthalmology one of the most successful specialties in providing quality care and patient satisfaction."

However, Dr. Pusin does see disadvantages to the advances in technology. "There is a down side to this success. The sophisticated and yet patient-friendly surgical techniques have lulled the public, legislators, and HCFA [Health Care Financing Administration]

into trivializing the skill, diligence, stress, and demands of being an ophthalmologist. Optometrists want to provide postoperative care, prescribe drugs, and even perform laser procedures. This impacts on the quality of care now and in the future.”

Eve Higginbotham, M.D., University of Maryland professor and chair of the ophthalmology department, and a member of the Baltimore City Medical Society’s Board of Directors, also believes the advances in medical and laser treatments for eye diseases have made major changes possible. The identification of genetic markers for a specific glaucoma is certainly encouraging. Our approach to management of patients at risk for glaucoma will be influenced by these discoveries in the 21st century. “There are new medications for glaucoma that are easier for patients to tolerate and new ways of doing glaucoma surgery which are more successful.”

In addition to the advances of medicine through new methods and technology, Dr. Higginbotham also sees managed care as having a significant impact on the medical world. “As a physician, I sometimes feel as if I am a small cog in a massive engine otherwise known as a contract. It is not known from one visit to the next if I will be seeing this same patient again. Glaucoma is a chronic disease. I may be in the midst of finalizing a patient’s medical regimen and the contractor may call the patient to tell her or him to see another doctor next time. The doctor-patient relationship has been severely impacted by managed care. However, somehow we must retain the very essence of medicine — the special relationships between doctors and patients,” explains Dr. Higginbotham.

Dr. Benkert agrees that managed care has impacted medicine the most over the last several years. “We are now living through the dismantling of American medicine as we have known it, the object of corporate takeover. We are moving toward a two-tier system of health care where those who can afford it have health care advantages unavailable to others.”

Dr. Smorzaniuk’s response also echoes what the other physicians expressed. “I can’t imagine anyone saying anything but managed care! I don’t think I ever heard the expression ‘cost-effectiveness’ while attending medical school in the late-70s. In the long run, this will have a positive impact — it has clearly demonstrated how gluttonous and wasteful our health care

system has become. Unfortunately, we are in the thick of a managed care feeding frenzy, with many of these companies skimming 10% to 30% off the top of the premium dollar as pure profit. I am optimistic in believing this is self-limited. The increasing bad press about HMOs and the awakening of businesses paying out large sums for health care will, eventually, bring their aggressive growth to an end.”

Dr. Winchell also concurs that managed care has had major effects on the health care industry, negative as well as positive. She describes her introduction to managed care as less than pleasant, “kicking, screaming, and retching; I was dragged up to speed.” Dr. Winchell now asserts that “in a ‘mature’ system, capitation of a population results in the physician taking the larger view and emphasizing prevention and health maintenance rather than treatment of illness. Since women physicians are over-represented in the primary care arena, this gives women a unique opportunity to assume the helm of the medical care in this country.”

How can organized medicine more effectively represent us?

The third question posed was “How can organized medicine (component, state or national societies) most effectively serve and represent physicians?” Lobbying government for the regulation of managed care, education, reaffirmation of medicine’s mission, and communication rounded out the solutions for more effective representation of Maryland’s physicians.

Dr. Higginbotham has several ideas for more effective representation. She believes organized medicine should lobby legislators to create regulations for the governance of managed care contracts. Dr. Higginbotham also thinks medical societies should assist physicians by providing leadership conferences. “One of the most important characteristics of an effective leader is to be a ‘people person,’ a characteristic which comes naturally to most physicians. It is the other skills which need honing, such as negotiating and quality management.”

Redefining and/or reaffirming the mission statement is another area organized medicine should address, according to Dr. Pusin. “Med Chi must be first and foremost a patient and physician advocate. It cannot be torn apart by individual agendas. Without defined goals,

organized medicine cannot speak with a single voice. We cannot be proactive in the future direction of medicine if government and third-party payers continue to divide and conquer us. Our membership will decline and there will be no voice for physicians."

Dr. Lee-Llacer allows that communication is the key to the competent representation of physicians around the state. "Close communication with members will keep the medical society apprised of member concerns. The concerns can be voiced in the House of Delegates and legislative meetings for lobbying issues, strategic planning, and for future direction of the medical society."

Dr. Smorzaniuk agrees that organized medicine should work toward better communication; however, she believes it should be with regard to the government and public. "Organized medicine should be a consistent, persistent, and respected voice to the legislature, at all levels of government," according to Dr. Smorzaniuk. "Organized medicine should take public stands on all issues that impact public health, be it block grants, smoking ordinances, gun control laws or clean air acts. It should work at reinstating the public image of physicians as patient advocates rather than adversaries."

Advice to future physicians

Our final question was "What advice or insights can you provide to students who are contemplating a career in medicine?" The variety of responses ranged from philosophical to humorous to optimistic.

In her search for philosophical insight to the undertaking of the medical profession, Dr. Snow believes "only people who care to serve their fellow man should undertake a medical career." She also adds, "I have always assumed that I (or anyone else) could accomplish whatever goal I had in mind if I worked hard enough to achieve it."

Dr. Winchell offers her own experience when she was deciding her future profession. "When I was a teenager I spoke with a physician about my interest in medicine as a career, and he (of course it was a 'he' — who knew any women physicians in the 50s?) lamented that medicine had so changed since he had become a physician that he didn't think it was a field worth going into anymore," recalls Dr. Winchell. "As I hear myself and my colleagues intoning the same lament, I conclude the 'Golden Age of Medicine' is whenever you

enter the process. Medicine is always changing and the delivery of health care is always evolving. Those students of medicine who are coming out of medical school today will be managed-care oriented, computer-oriented, and bureaucracy-tolerant compared to my generation of physicians. One thing about medicine that will never change is physicians have the privilege of becoming an important and intimate participant in hundreds of lives in the course of their careers. We have the opportunity to affect people's lives and we do every day through the simple things we say to guide our patients. Now, that's a turn-on few careers offer!"

Allowing that there are many positives to entering the medical profession, several of the physicians interviewed lent a more dubious look at medicine as a career. "It's stressful, but there are still rewards, not all financial," replied Dr. Farrell. "Only by putting patients first can we retain pride in our profession." In conclusion she advises "never sign a 'gag rule' contract."

Dr. Pusin agrees that a lot has changed, but there are still rewards. "For students contemplating a career in medicine, there is much more economic uncertainty and much less independence and freedom of choice. At the present, some days are so filled with hassles one often asks, 'What am I doing here?' Fortunately, the good days still outnumber the bad. The rewards of making someone better and helping society are like birdying the 18th hole — you want to return tomorrow to try again." Finally, Dr. Smorzaniuk offers students a more pessimistic view, "if you can think of anything else to do, do it. [Sorry so cynical]."

Our final response, provided by Dr. Benkert, is more optimistic. "I believe medicine is a vocation and it has been a most rewarding and satisfying career for me. My son will be following that path as he began medical school in September 1996. He will practice medicine very differently than I did for most of my career. But my son and the other young people joining him in preparation for their medical careers are bright and dedicated individuals. I believe they will adapt and be imaginative and innovative in their approach to medicine."

BEVERLY COLLINS, M.D., M.B.A.

Dr. Collins is a postdoctoral fellow in the department of epidemiology and preventive medicine at the University of Maryland at Baltimore. ■ [USA]

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Manifesto for a New Medicine: Your Guide to Healing Partnerships and the Wise Use of Alternative Therapies. James S. Gordon, M.D. Addison Wesley Publishing Company, Inc. 1996. 358 pages.

While this book lives it up to its title, it is little more than a “public declaration of views” and falls far short of achieving its subtitle. Although written by a psychiatrist with excellent credentials, the book is seriously lacking in useful information for the medical community. The author discusses how he became interested in alternative medicine, the failings of “biomedical medicine,” and the history of many alternative therapies, interspersing case histories from his practice. Unfortunately, he makes no attempt to explore the therapies in detail, nor to suggest how and when each might be of benefit.

indexed to serve as a reference book. The author also does not attempt to furnish any evidence of effectiveness other than anecdotes, despite his advisory position with the National Institute of Health's (NIH) Office of Alternative Medicine. Those with an interest in alternative medicine may be left feeling unsatisfied. Those who are curious will, no doubt, be left shaking their heads. Perhaps it was written for a lay audience, but I don't think it'd be particularly useful for them either.

RICHARD C. MOORE, MD, MPH
Dr. Moore is a family physician in
Danville, Virginia. ■

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Check Us Out Today!

The CMERC Update, now in its second year, informs all Med Chi accredited CME sponsors about the activities of the Continuing Medical Education Review Committee (CMERC). The CMERC has received feedback (both negative and positive) from our accredited sponsors this past year. This exchange of information has kept us all better informed.

CMERC awarded ACCME's Rutledge W. Howard Award

On August 18, 1997, the Accreditation Council for Continuing Medical Education (ACCME) Committee for Review and Recognition notified Med Chi's Continuing Medical Education Review Committee (CMERC) of its selection to receive the 1997 Rutledge W. Howard Award for Innovation and Creativity in the Accreditation Program. The award consists of a statuette and a check.

Med Chi president Thomas E. Allen, M.D., wrote a letter to committee chair Duesdedit Jolbitado, M.D., commending the committee and staff for this recognition from a national organization. Committee member Barbara Hulfish, M.D., who represented the CMERC at the ACCME's State Medical Society Meeting in Chicago, accepted the award.

Three CME sponsors reaccredited

At its September meeting, the CMERC reaccredited three CME sponsors: Springfield Hospital Center of Sykesville, Maryland, Francesco DiLeo, M.D., chair; Spring Grove Hospital Center of Catonsville, Maryland, Kripa Kashyap, M.D., chair; and Prince George's Hospital Center of Cheverly, Maryland, Haluk Boneval, M.D., chair.

Other actions by the CMERC

The committee also conducted preliminary reviews of three reaccreditation applications and reviewed four interim reports. Two sponsors voluntarily withdrew from accreditation, although one expects to reapply after further preparation.

Interim reports

An interim report is documentation required of accredited continuing medical education sponsors during the accreditation period. The CMERC, as the accrediting body, uses the interim report to identify problems or weaknesses in the sponsor's CME program. Ideally, problem and weak areas of the program can then be improved prior to the sponsor's next reaccreditation survey. After careful review of the interim report, the committee votes on what action to take: it may accept the interim report; it may request more information or documentation; or it may schedule a consultation or a site visit. On occasion, the CMERC has voted to change a sponsor's accreditation status from full accreditation to pro-

bationary accreditation on the basis of an interim report that identifies serious problems and/or fails to demonstrate improvement in a CME program. Because they can affect a sponsor's accreditation status, the CMERC hopes that sponsors take these reports seriously.

Current CMERC agenda

A subcommittee met with staff in September to begin updating the *CMERC Handbook*. In addition, staff continues to correspond with accredited CME sponsors, schedule surveys, and preview applications for completeness before committee members review them.

At the September meeting, committee members reviewed a list of the CMERC's accomplishments during the past two years and a list of planned activities for the coming year. Among these, recruiting new committee members and training site surveyors are two of the most important activities. New members receive training by accompanying experienced members on site visits. CMERC members may approach other members of Med Chi who have an interest in continuing medical education and who have the time to devote to committee activities.

New statement from the ACCME

The ACCME has developed the following new statement for immediate use on all printed materials for jointly sponsored activities:

This activity has been planned and implemented in accordance with the essentials and standards of the accreditation by the Continuing Medical Education Review Committee of Med Chi through the joint sponsorship of (insert name of Med Chi accredited sponsor) and (insert name of non-accredited sponsor). The (insert name of Med Chi accredited sponsor) is accredited by the Medical and Chirurgical Faculty of Maryland's CMERC to provide continuing medical education for physicians.

DEUSEDIT L. JOLBITADO, M.D.

Dr. Jolbitado is the chair of the Continuing Medical Education Review Committee (CMERC) of the Medical and Chirurgical Faculty of Maryland. ■

The purpose of this newsletter is to inform CME chairpersons, committee members, and all interested physicians about the activities of the CMERC and about the policies and procedures that affect the implementation of CME programs in Maryland. When appropriate, news from the ACCME is included.

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November/December, 1997

New Recommendations for Screening Infants and Children for Tuberculosis

National and state guidelines for tuberculosis screening of children have changed recently, dramatically reducing the number of children who should be screened. These recommendations include:

1. Screen only children at high risk for tuberculosis; avoid routine screening of low risk children.
2. Use only the Mantoux test; discontinue use of multiple puncture tests (MPT's). Tests should be read by a trained health care worker.
3. Mantoux results should be interpreted based on TB risk factors.
4. History of BCG vaccination is not a contraindication to TB skin testing.
5. Provide 9 months of preventive therapy to TB infected children up to age 21.
6. Conduct associate investigations (search for the source case) for TB infected children under age 6.

The rationale for the above recommendations are discussed below.

1. Screen only children at high risk for tuberculosis; avoid routine screening of low risk children (Table 2).

Jeffery Starke, M.D., a national expert on pediatric tuberculosis, succinctly states the argument against

routine screening of low risk children. "There are only two ways in which a child in the United States can be infected with *M. tuberculosis*: the child can be infected in a foreign country and bring latent tuberculosis infection with him or her during immigration, or the child can be infected in the United States by a contagious adolescent or adult.... The central activity for the prevention of childhood tuberculosis is the contact investigation" of TB cases rather than routine screening.¹

In 1994 the American Academy of Pediatrics (AAP) recommended an end to routine annual testing of low risk children.² The AAP recommendations were further clarified in 1996 and include the following general principles:

- Test only children at increased risk of TB exposure;
- Routine testing in low prevalence communities is not indicated;
- Test HIV infected children annually;
- Consider screening children from high prevalence regions or with incomplete or unreliable histories for risk factors at ages 4 to 6 and 11 to 16 based upon local epidemiology and regional TB control recommendations.³

The rate of tuberculosis in Maryland children age 0-14 has remained low and stable for the past decade, ranging from 1.4 to 2.7 per 100,000 (16 to 28 cases per year).⁴ While recent TB screening data on

Table 1. Prevalence of TB Infection in Selected Groups of Maryland Children

Location	Group	Year	# Positive/ # Tested	% Positive
Students in Baltimore City from Elementary School A ⁵	Contacts to TB Case (5 mm)	1995	2 / 13	15.4
	Other Children (10 mm)		4 / 576	0.7
Baltimore City ⁶	Inner City Pediatric Clinic	1994	5 / 573	0.8
MD Juvenile Services ⁷	Incarcerated Adolescents	1993	23 / 1,326	1.7
Frederick County Middle/High School A ⁸	Contacts to TB Case (5 mm)	1995	3/122	2.5
	Other Students (5 mm)		34 / 1,804	1.9
Montgomery County ⁹	Foreign Born School Children	1993	401 / 2,980	13.9
		1994	471 / 2,884	16.3

Maryland children is limited, all available population-based data indicates low prevalence of TB infection (see Table 1), with the exception of foreign born children. Perhaps most noteworthy is the study by Serwint et. al., of an indigent, Baltimore inner city clinic population in which only 0.8% (5/573) of children tested were skin test positive.⁶ In contrast, foreign born students, who constitute a known high risk group and who are routinely screened upon school entry into the Montgomery County school system, have a significantly higher prevalence of skin test positivity, 13.9% (1993) and 16.3% (1994).⁹

The Maryland Pediatric TB Expert Panel¹⁰, which met in June 1997 to review the AAP guidelines in light of available prevalence data, concluded that **no geographic area of Maryland constitutes a "high prevalence" region warranting routine tuberculin testing of all children.** Testing should instead be targeted to children with individual TB risk factors. The Expert Panel developed Maryland specific screening guidelines based upon AAP and statewide epidemiological data (see Table 2).¹¹ These guidelines have been adopted by the Maryland Healthy Kids Program for children who are Medicaid recipients.

2. Use only the Mantoux test; discontinue use of multiple puncture tests (MPT's). Tests should be read by a trained health care worker.

In 1994, the Committee on Infectious Diseases of the AAP recommended the use of the Mantoux PPD test instead of multiple puncture tests (MPT's) because of better sensitivity and specificity of the

former. The Mantoux test requires reading by palpation by a trained health care worker (not parents or guardians).¹ Results should always be recorded in millimeters; if there is no reaction it should be recorded as 0 mm.

The advantages of targeting Mantoux testing to high risk children are significant. Testing of low risk children produces false positive results leading to unnecessary concern and exposure of uninfected children to risks associated with preventive chemotherapy. Selective testing of children at higher risk focuses time, energy, and resources on the population that will most benefit from preventive treatment. It increases the incentive to promote return visits for test reading and follow-up.

3. Interpret results of Mantoux tests based on TB risk factors.

A Mantoux test result is considered positive at three different levels (≥ 5 mm, ≥ 10 mm, and ≥ 15 mm) based upon risk factors for TB infection and risks for development of disease given TB infection (see Table 3).¹¹ Questions about interpretation of skin test results can be referred to Maryland local health departments.

4. History of BCG vaccination is not a contraindication to TB skin testing.

Most foreign countries still use BCG as part of their TB control programs, especially for infants. In persons vaccinated with BCG, sensitivity to tuberculin and subsequent protection from disease is highly variable. Reaction sizes tend to be smaller than those related to tuberculosis infection. The

Table 2. 1997 Maryland Tuberculin Skin Test Recommendations for Children

Test Immediately	<ul style="list-style-type: none">• Contacts of persons with confirmed or suspected tuberculosis.• Children with radiographic or clinical findings suggesting TB.• Children ≥ 6 months of age who have immigrated from, or have lived ≥ 12 months in countries where TB is endemic*.• Children ≥ 6 months of age who are likely to require immunosuppressive therapy (prior to initiating therapy).• Foster children ≥ 6 months of age upon entry into the foster care system.
Test annually	<ul style="list-style-type: none">• Children infected with HIV.• Incarcerated adolescents (test on admission and annually while incarcerated).
Test every 2-3 years	<ul style="list-style-type: none">• Children living in households with adults who are HIV- infected.• Children who are members of migrant farm worker families.
Upon school system entry	<ul style="list-style-type: none">• Foreign born children of any age from countries where TB is endemic* who do not have prior Mantoux tuberculin skin test result documented in the U.S.

Table 3. Definition of a Positive Mantoux Tuberculin Skin Test in Children

Reactions ≥ 5 mm
<ul style="list-style-type: none">• Children less than 1 year of age (Maryland specific guideline)• Children in close contact with known or suspected cases of tuberculosis.• Children suspected of having tuberculosis disease.• Children with immunosuppressive conditions, including HIV infection .• Children who are receiving long-term corticosteroid therapy.
Reactions ≥ 10 mm
<ul style="list-style-type: none">• Children between the ages of one and four years.• Children with other medical risk factors (e.g., diabetes mellitus, lymphoma, chronic renal failure, being 10% or more below ideal body weight, leukemias and other malignancies).• Children with increased environmental exposure including:<ul style="list-style-type: none">- those born in, or whose parents were born in a foreign country where TB is endemic*.- those who have lived ≥ 12 months in a country where TB is endemic*.- those living in households with adults who are HIV- infected.- those who are members of migrant farm worker families.
Reactions ≥ 15 mm
<ul style="list-style-type: none">• Children > 4 years of age with no known risk factors.

Recommendations of the Maryland Pediatric TB Expert Panel based upon the American Academy of Pediatric Guidelines and Maryland TB epidemiology.

- * Areas of the world where tuberculosis is considered endemic include Africa, Asia, Latin America, the Middle East, and some areas of the former Soviet Union and Eastern European block countries where the public health infrastructure has been disrupted.

AAP and Maryland TB Control Program do not consider a history of BCG vaccination to be a contraindication to tuberculin skin testing. A reaction to tuberculin is more likely due to infection with *M. tuberculosis* if:

- the induration is large;
- the person was vaccinated long ago;
- the person is a recent contact of a person with infectious TB;
- there is a family history of TB;
- the person comes from an area where TB is common; or
- the chest radiograph findings show evidence of previous TB.

In a BCG-vaccinated person who has any of the preceding risk factors, a positive tuberculin reaction probably indicates infection with *M. tuberculosis*. Such persons should be evaluated for isoniazid preventive therapy after disease has been ruled out.^{12.}

5. Provide 9 months of preventive therapy to TB infected children up to age 21.

Preventive therapy substantially reduces the risk of tuberculosis disease. The Maryland Expert Panel recommends that 9 months of preventive therapy be given to children, adolescents and young adults (up to age 21). Isoniazid is given daily, 10 mg/kg (maximum, 300 mg), in a single dose. Twice a week directly observed therapy (observation by a trained health care worker of the child taking each dose) can be utilized to assure adherence. Each dose of isoniazid in the twice-a-week regimen is 20 to 30 mg/kg (maximum, 900 mg).¹³

6. Conduct associate investigations in TB infected children under age 6.

An associate investigation should be conducted for children under age 6 with tuberculosis disease or infection. The purpose of this investigation is to search for the source of the child's TB infection.¹³ Maryland local health departments will assist with associate investigations.

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| Type II diabetes: Current concepts in management , sponsored by The Johns Hopkins University School of Medicine. Credits: 7.5 Cat 1 AMA credits. Fee: \$50/physicians; \$35/residents, fellows, allied health professionals. | Nov. 15 |
| AIDS managed care , sponsored by The Johns Hopkins Medical Institutions, Kaiser Family Foundation, Infectious Diseases Society of America, and Health Resources and Services Administration, at Renaissance Harborplace Hotel, Baltimore. Fee: \$300. | Nov. 17-18 |
| Women's health conference , sponsored by The Johns Hopkins Medical Institutions, department of gynecology and obstetrics, at Sheraton Baltimore North Hotel, Towson. Credits: 7 Cat 1 AMA credits. Fee: \$95/physicians; \$75/residents, fellows, allied health professionals. | Nov. 21 |
| Investigating and organizing clinical research , sponsored by The Johns Hopkins Medical Institutions, department of medicine. Credits: 8 Cat 1 AMA credits. Fee: \$250/physicians; \$195/residents, fellows, allied health professionals. | Nov. 21 |
| Eighth annual neurology for the primary practitioner , sponsored by The Johns Hopkins Medical Institutions, department of neurology, at Harbor Court Hotel, Baltimore. Fee: \$140/physicians; \$90/residents, fellows, allied health professionals. | Dec. 6 |
| Topics in ambulatory medicine , sponsored by The Johns Hopkins University School of Medicine and The Johns Hopkins Bayview Medical Center, at the Renaissance Harborplace Hotel, Baltimore. Fee: \$550/physicians; \$325/residents, fellows, allied health professionals. | Dec. 10-12 |

Continuously throughout the year

- Visiting preceptorship in pediatric critical care medicine.** Ongoing five-day preceptorship by appointment. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$600.
- The department of radiology and radiological sciences** offers several courses in abdominal and obstetrical ultrasound. Info: P. Williams, 410-955-3169.
- Visiting physicians.** Offered throughout the year for experience in the lab and participation in read-in sessions; by appointment only. Credits: 40 Cat 1 AMA/PRA credits. Fee: \$500.
- Johns Hopkins medical grand rounds.** Accredited audiovisual continuing education series of case discussions for clinicians; 30 topics per year in five bimonthly programs. Individual and group subscriptions. Credits: 40 Cat 1 AMA/PRA credits. Info: 410-955-3988.
- Johns Hopkins sports medicine grand rounds.** Accredited continuing education series of presentations on sports medicine topics applicable to primary care and orthopaedic surgery. Discussions first Thursday of each month. Info: Amy Taylor, 410-383-0600.

University of Maryland School of Medicine

For each course, additional information may be obtained by contacting the Program of Continuing Education, University of Maryland School of Medicine, Room 14-011, BRB, 655 W. Baltimore St., Baltimore, MD 21201 (410-706-3956), or by calling the phone number listed after a specific program. Fax 410-706-3103.

Self-Directed CME Activities

- CD-ROM-based interactive multimedia radiology teaching file for Mac or PC w/single-user licenses (SUL), site licenses (SL), or multisite licenses (MSL).** Credits: 60 Cat 1 AMA credits. Expires 9/98. Fee: \$149/SUL, \$395/SL, \$595/MSL. Info: Lorraine Smith, 410-528-8502.

University of Maryland School of Medicine (continued)

Academic rounds and conference. Each academic department within the school of medicine has a series of lectures and/or seminars available to physicians. Cat 1 AMA credits available.

Miscellaneous

Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at North Arundel Hospital. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.	Nov. 20
Clinical breast examinations using mammapare technique – a practicum for physicians , sponsored by the Medical and Chirurgical Faculty of Maryland, at Howard County General Hospital, Columbia. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	Nov. 25
Cancer prevention in community practice , sponsored by the Medical and Chirurgical Faculty of Maryland, at Prince George's Hospital Center. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056.	Dec. 3
Surveillance of vaccine-preventable diseases , presented via satellite by the Centers for Disease Control and Prevention, hosted by the Center for Immunization, Maryland Department of Health and Mental Hygiene. Credits: CMEs available. Fee: none. Info: Sandra Kash, 410-767-6679.	Dec. 4
Rural Health Conference, Communities leading the way , sponsored by The Maryland Academy of Family Physicians (MAFP), at Loews Annapolis Hotel, Annapolis, Maryland. Credits: 17 AAFP prescribed credits. Fee: \$125. Info: 410-747-1980.	Dec. 4–6
25th annual Williamsburg conference on heart disease , sponsored by the American College of Cardiology at Williamsburg Lodge, Williamsburg, Virginia. Credits: 19 Cat 1 AMA. Info: 800-253-4636, ext. 695, Fax 301-897-9745.	Dec. 7–10
Clinical breast examinations using mammapare technique – a practicum for physicians , sponsored by the Medical and Chirurgical Faculty of Maryland, at Northwest Hospital Center, Randallstown. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	Dec. 10
14th annual CME clinical update in pulmonary medicine , sponsored by the department of pulmonary medicine, Deborah Heart & Lung Center, Browns Mills, NJ, at the Trump World's Fair Casino, Atlantic City, New Jersey. Credits: 7 Cat 1 AMA. Fee: \$175/physicians; \$100/allied health professionals, physicians-in-training (until Oct. 14); \$225 and \$130, respectively, after Oct. 14. Info: 201-385-8080, Fax 201-385-5650 (e-mail: jrosenberg@cbcbiomed.com).	Dec. 13
Aspen radiology review course: what you need to know “in the snow,” is sponsored by the University of Florida, College of Medicine, at the Ritz-Carlton Resort Hotel, Aspen, Colorado. Credits: 25 Cat 1 AMA credits. Fee: \$625/physicians; \$495/residents, fellows, technologists, full-time military. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com).	Jan. 7–11
Clinical breast examinations using mammapare technique – a practicum for physicians , sponsored by the Medical and Chirurgical Faculty of Maryland, at Anne Arundel Medical Center, Annapolis. Info: Jade Leung, 410-539-0872 or 1-800-492-1056.	Jan. 14
Spiritual wisdom and the practice of psychotherapy , sponsored by Sheppard Pratt Health System, at the Conference Center at Sheppard Pratt, Baltimore. Info: Barbara Johnson, professional education programs, Sheppard Pratt, 6501 N. Charles St., Baltimore, MD 21285-6815, 410-938-4598, Fax 410-938-4596.	Jan. 16–17

Miscellaneous (continued)

- Breast imaging today and tomorrow**, is sponsored by the International Institute for Continuing Medical Education, at The Breakers Resort Hotel, Palm Beach, Florida. Credits: 25.25 Cat 1 AMA credits. Fee: \$650/physicians; \$400/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Jan. 19-23**
- 4th annual neuroradiology: a comprehensive review**, is sponsored by the University of California, San Diego, at The Ritz-Carlton Hotel, Palm Beach, Florida. Credits: 24 Cat 1 AMA credits. Fee: \$650/physicians; \$425/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Jan. 19-23**
- Clinical breast examinations using mammacare technique – a practicum for physicians**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Potomac Physicians, P.A., Baltimore. Info: Jade Leung, 410-539-0872 or 1-800-492-1056. **Jan. 21**
- 6th annual musculoskeletal MR course**, is sponsored by the University of California, San Diego, at The Breakers Resort Hotel, Palm Beach, Florida. Credits: 22.5 Cat 1 AMA credits. Fee: \$595/physicians; \$350/residents, fellows, technologists. Info: Ryals & Associates, Inc., 770-641-9773, Fax 770-552-9859 (e-mail: webmaster@ryalsmeet.com). **Jan. 26-30**
- Cancer prevention in community practice**, sponsored by the Medical and Chirurgical Faculty of Maryland, at Northwest Hospital Center, Baltimore County. Free CME credits available. Info: Carol Schwartz, 410-539-0872 or 1-800-492-1056. **Jan. 28**

Continuously throughout the year

- Fluorescein angiography conference**, sponsored by the Retina Center of Maryland, Baltimore. First and third Mondays of each month; 8:00 a.m. – 9:00 a.m. Fee: none. Info: C. Love, 410-337-4500.
- Sinai Hospital of Baltimore medical grand rounds**, Zamoiski Auditorium, Thursdays, 9:00 a.m. – 10:00 a.m. Info: 410-578-5528.



PHYSICIAN'S RECOGNITION AWARD

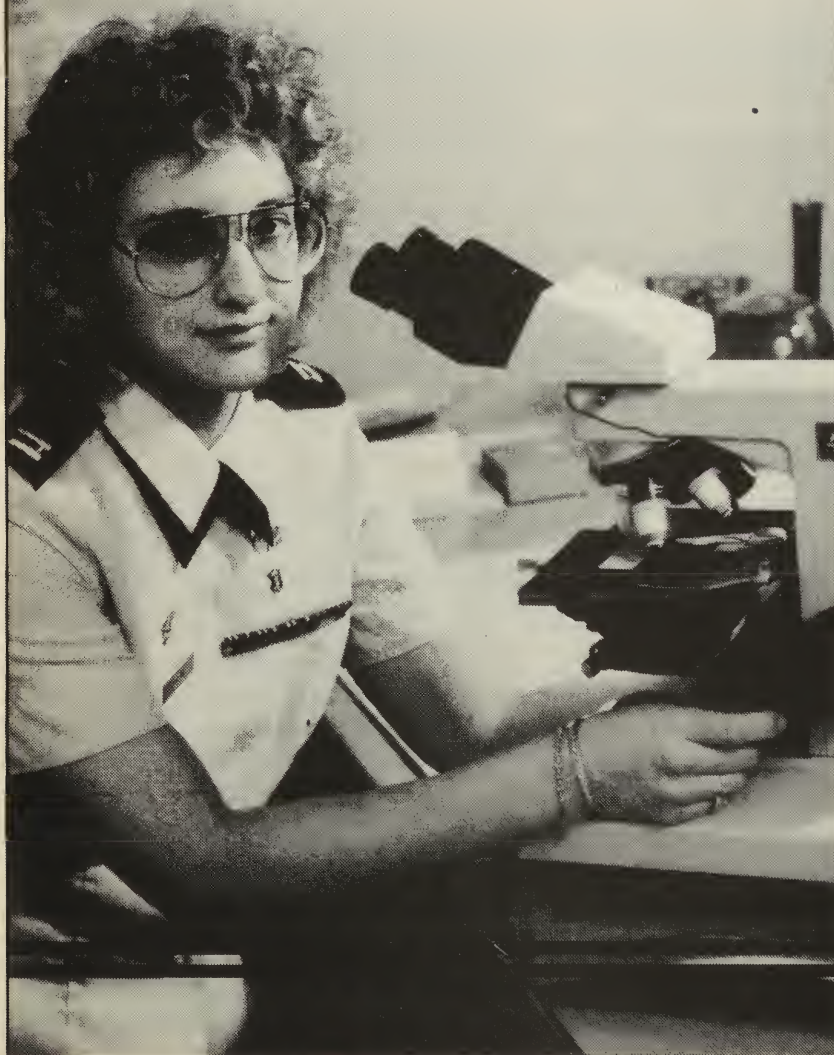
During August and September 1997, the physicians listed below received the American Medical Association (AMA) Physician's Recognition Award. Established in 1968, the award's purpose is to encourage physician participation in continuing medical education and to recognize those physicians who have voluntarily completed programs of continuing medical education.

Carol Ann Blank
Leonard Dean Cutler
Aaron B. Daugharthy
Charles Hawes Evans
Oscar A. Farias
Jay Myron Grodin
Isis S. Hannallah
Alvanus Howland Hartley
Mark Jonathan Jaffe
Allen Herbert Judman
Stavros I. Karakozis

Sitaramamma Kottapalli
Richard Collison Lang
Raul A. Lazarte
Jeffrey Mark Levitt
Lawrence Scott Linder
Rolando Mascardo
Ashwin I. Mehta
Saverio Mirarchi
Julio Cesar Novoa
Victor Manuel Pineiro

Craig Chas Porter
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- Spouses of deceased members are entitled to two complimentary insertions for the disposal of the deceased physician's practice or equipment.
- Box numbers are provided free of charge.
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Box replies, advertising copy, and prepayments should be sent to:

Vivian Smith, MMJ, 1211 Cathedral St.,
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For more information, call Vivian Smith at
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PHYSICIAN PLACEMENT SERVICE

The Medical and Chirurgical Faculty of Maryland maintains a Placement Service for the convenience of Maryland physicians, hospitals, and communities in search of candidates for positions available in our state. A detailed description of such opportunities should be forwarded to:

Physician Placement Service, 1211 Cathedral Street, Baltimore, MD 21201

410-539-0872 • 1-800-492-1056

Physicians wishing to locate in Maryland are invited to submit a résumé to be kept on file with the Physician Placement Service. Candidates are requested to inform the Faculty when they are no longer available for consideration opportunities in Maryland.

MMJ announcements for physician placements in the classified advertisements are charged at the regular classified advertising rate.

Cite SEER

Attendees of a Centers for Disease Control (CDC) multistate workshop on the public health issues about *Pfiesteria piscicida* determined a combined set of environmental conditions and clinical signs and symptoms that together may represent adverse consequences of exposure to the organisms (*MMWR Morb Mortal Wkly Rep* 1997;46:951-952). The environmental conditions are exposure to estuarine water characterized by any of the following: fish with lesions consistent with *P. piscicida* or morphologically related organisms (MRO) toxicity (20% of a sample of at least 50 fish of one species having lesions); a fish kill involving fish with lesions consistent with *P. piscicida* or MRO toxicity; or a fish kill involving fish without lesions, if *P. piscicida* or MROs are present and there is no alternative reason for the fish kill. The clinical features in humans include any of the following: memory loss, confusion, acute skin burning (on direct contact with water), three or more of an additional set of conditions (headaches, skin rash, eye irritation, upper respiratory irritation, muscle cramps, and gastrointestinal complaints [i.e., nausea, vomiting, diarrhea, and/or abdominal cramps]). Public health activities were recommended for this emerging environmental and potential occupational health issue.

Is raising your grandchild bad for your health? Results of a National Longitudinal Study suggest that grandparents who serve as the primary caregiver of a grandchild have an increased level of depression (*Arch Fam Med* 1997;6:445-452). While the authors do not state the act of raising grandchildren is in itself a factor for depression, they note that it is an indication of changes in familial roles and life circumstances. Because the study found that grandparents who provide primary care for a grandchild are almost twice as likely to experience depression, physicians are encouraged to pay attention to the most at-risk grandparent caregivers: new caregivers, those in poor health, and younger women caregivers.


The first report of known cases of poisonings in the United States resulting from the illegal use of aldicarb as a commercially prepared rodenticide was recently published. (*MMWR Morb Mortal Wkly Rep* 1997;46:961-963). Although registered as a pesticide for use against insects, mites, and nematodes on field crops, certain vegetables and fruits, and ornamental plants in the United States, aldicarb is not registered for use as a rodenticide and the Environmental Protection Agency classifies it in its highest toxicity category. Tres Pasitos, the aldicarb indicated in both cases reported in this study, is legal and widely used as a rodenticide in the Dominican Republic. The poisoning cases have occurred primarily among emigrants from the Dominican Republic and community outreach efforts are underway in New York City where Tres Pasitos is often sold in stores. Physicians suspecting exposure to this rodenticide product should contact their local health departments, especially in areas that include emigrant populations from the Dominican Republic.

Whooping cough (pertussis) incidence continues to rise despite widespread vaccination and effective therapy (*Am Fam Phys* 1997;56:1121-1128). **Pertussis, a reportable disease since 1922, is considered highly contagious and infection in infants and young children are potentially life-threatening.** Authors of this report indicate that although many have been vaccinated against this illness, the benefits of the vaccination decline steadily and provide almost no immunity after 10 to 12 years. They therefore suggest that the best way to decrease pertussis infection may be to develop a more effective primary childhood vaccination. The authors discuss primary prevention and outbreak control.

An investigative study of 124 ambulatory elderly patients who discontinued medications found that adverse drug withdrawal events (ADWEs) occurred in almost one third of this population. Cardiovascular and central nervous system drugs were most frequently associated with ADWEs. However, it was noted that most (74%), of the medications can be stopped without causing ADWE. Because ADWE resulted in hospitalization or emergency care 36% of the time, authors feel that **physicians need to exercise caution in discontinuing medications in elderly patients** (*Arch Intern Med* 1997;157:2205-2210). ■

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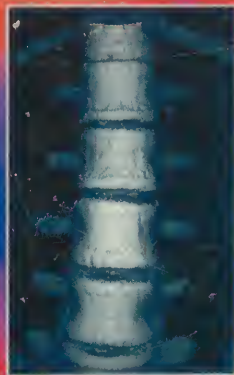
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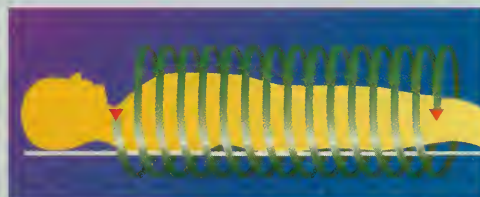
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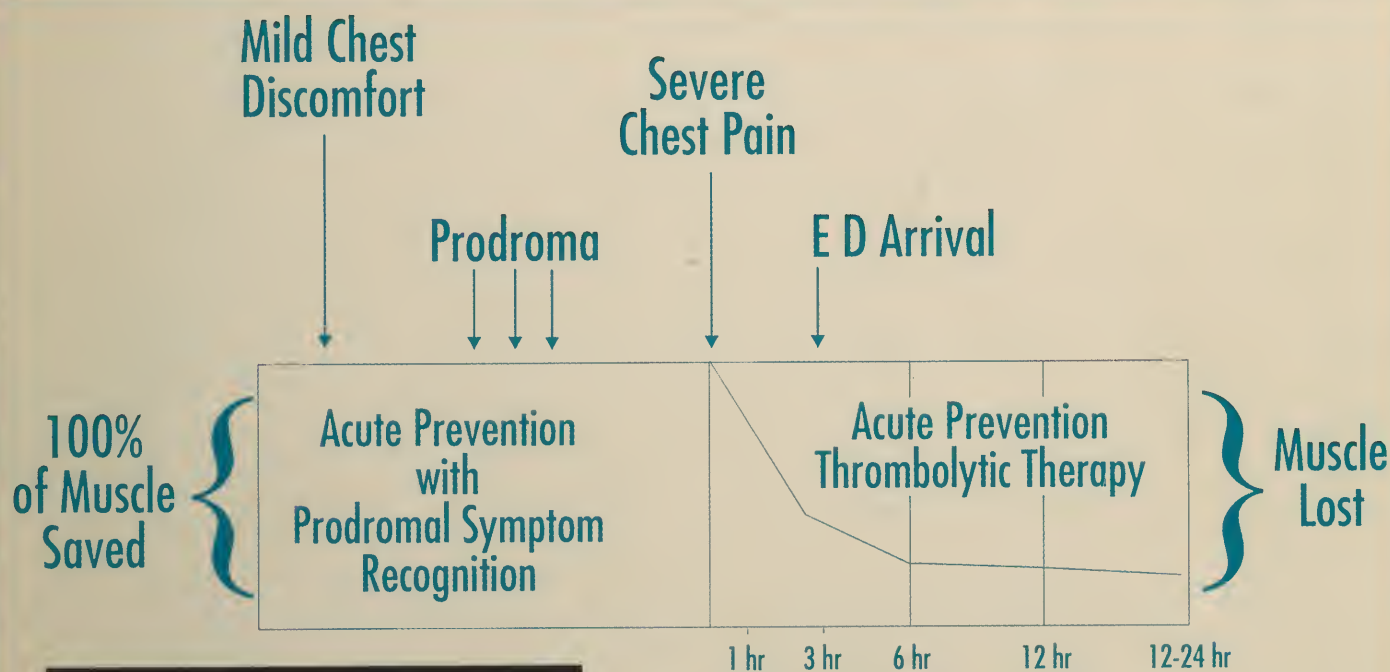
Chest Pain Units

(in Emergency Departments)

in the War Against Heart Attacks

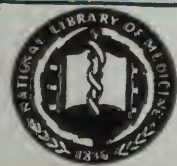
PROCEEDINGS FROM THE FIRST MARYLAND CHEST PAIN CENTER RESEARCH CONFERENCE

Guest Editor: Raymond D. Bahr, M.D., F.A.C.P., F.A.C.C



THE PRODROMAL OFFENSIVE

The Maryland scene . . .



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The Strategy of Chest Pain Units (in Emergency Departments) in The War Against Heart Attacks

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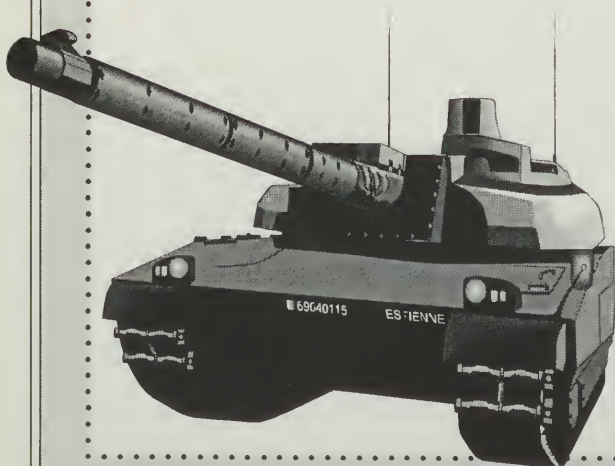
The beaming sword shall never rust
Maryland, my Maryland

Remember CPR's initial thrust
Remember Osler's bedside trust

and Early Awareness with the just
Maryland, my Maryland



The Maryland Initiative
The First Battalion



INTRODUCTION

Interpretive medicine applied to the heart attack problem in Maryland

The present state of heart attack care has many of its roots in Maryland. Perhaps the greatest discovery in heart attack care began in Baltimore — cardiopulmonary resuscitation (CPR). Drs. William Kouwenhoven and James Jude, from Johns Hopkins, discovered the use of chest compressions and the external defibrillator, and Dr. Peter Safar, from Baltimore City Hospital (now Johns Hopkins Bayview), discovered the effectiveness of mouth-to-mouth resuscitation. When combined, the discovery of these techniques led to the development of CPR. Also instrumental were Drs. Leonard Scherlis and Donald Dembo from University of Maryland in the rapid dissemination of the CPR information to the professional community. And, Captain McMahon, of the medical bureau of the Baltimore City Fire Department, quickly enhanced the emergency response system throughout Maryland.

It was the direct result of the successful practice of CPR and the knowledge that sudden and unexpected death took place in heart attack patients that Dr. Hughes Day, a general practitioner in Bethany, Kansas, conceived the idea of placing patients prone to the problem in an environment with people who knew what to do with this cardiac arrest complication. Thus the first coronary care unit was conceived. It rapidly brought together nurses and physicians in a remarkable partnership that improved the care of heart attack patients. Within seven years, every hospital in the United States had a coronary care unit. It was “an idea whose time had come.”

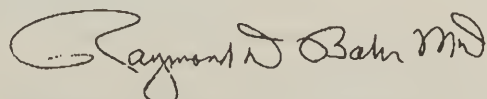
It made sense to proceed in that direction. Coronary care units became the research benches for heart attack care advances over the last 30 years and they have shown tremendous progress. Unfortunately, we seem to be plateauing and not reaching patients in the early stages of their attacks.

The purpose of this conference was to interpret another strategy to move us away from the crashing myocardial infarction patient and the need for thrombolytic therapy toward the early identification through prodromal symptom recognition to bring about acute prevention of the problem. Seen in this light, the occurrence of an acute myocardial infarction represents a failure to detect early symptoms, and the term myocardial infarction may be a better term than myocardial infarction.

The chest pain units strategy being developed in Maryland will still make available cardiopulmonary resuscitation and thrombolytic therapy for crashing acute myocardial infarction patients, but the major emphasis will be to make the chest pain unit be more friendly to patients with other forms of chest discomfort that harbor patients with myocardial ischemia. Organizing the emergency department to do so will give it the capability of properly evaluating patients and discharging most of them without unnecessary admission, thus opening the door to the community for more patients to be properly evaluated. This paradigm shift can only take place with proper evaluation in a prepared emergency department.

This conference brought together the latest information in chest pain centers carrying out this mission. The use of acute nuclear scanning and cardiac markers were highlighted for their role in this mission. Hopefully the information contained in this supplement will help you and your patients, as well as enlighten you to see a new approach that allows early intervention that brings about acute prevention of heart attack death and damage in Maryland's citizens. It also serves as a prototype challenge to other states to do likewise.

Sincerely,



RAYMOND BAHR, M.D., F.A.C.P., F.A.C.C.
Medical Director
The Paul Dudley White Coronary Care System
St. Agnes HealthCare



IN MEMORIAM

Robert Doherty, M.D.

by Robert Barish, M.D.,

*formerly director of the emergency department, University of Maryland,
now chief executive officer, UniversityCARE, University of Maryland*

Emergency medicine and the college lost a young leader when Robert Doherty, M.D., died of cancer on January 29, 1997, at the age of 35. Doherty was chief of emergency care services at the Veterans Affairs Medical Center in Baltimore and assistant professor at the University of Maryland School of Medicine, just prior to his death. He also served as director of education and residency coordinator at Mercy Medical Center in Baltimore.

Doherty made a number of significant contributions to ACEP during his short life. He cofounded the college's Young Physicians Section and served as president of EMRA from 1990 to 1991. "There was a lot going on when Bob assumed the presidency of EMRA," said Robert Suter, D.O., who served under Doherty as president-elect of EMRA. "We had a very close team-oriented board under Bob and he was instrumental in setting EMRA's financial plans and financial policies to secure the future of EMRA. This had great long-term implications for the organization. During these years, we transitioned from an association essentially supported by the generosity of other organizations to a self-sufficient organization with ongoing management by ACEP under a responsible business arrangement.

"Bob also instituted what he liked to call the 'EMRA information series,'" continued Suter. "These were handbooks on topics such as antibiotics and medical student information that provided a tangible benefit of membership to EMRA members. We welcomed Bob's leadership during these years and we all feel his loss now."

"He became president of EMRA when there was a need for direction," said Larry Alexander, M.D., a close friend and member of the EMRA board during Doherty's tenure. "Bob had a plan and a vision for the future, setting the groundwork for what EMRA has become."

Doherty made other contributions to medicine and emergency medicine, in particular. During his medical education, he founded the Georgetown University Emergency Response Medical Service (1980). He served on the Maryland Chapter's Board of Directors. And, he contributed to emergency medicine's academic credibility through his numerous research interests, including emergency cardiology, environmental emergencies, and EMS-related research. He was a current member of the peer review panel of the Pan American Journal of Trauma; from 1989 to 1991 he edited EMRA's newsletter *EM Resident* and was an abstract preparer for *Annals of Emergency Medicine* and the *Journal of Emergency Medicine* from 1988 to 1990. He was a frequent contributor of full chapters, articles, and invited speeches on his various research interests.

A recipient of many awards and commendations for his contributions, he won EMRA's Joseph F. Waeckerle, M.D., Founders Award in 1996 and was recognized by the college with a Resolution of Commendation at the 1996 Scientific Assembly in New Orleans.

Doherty received a Bachelor of Arts degree (Cum Laude) from Georgetown University in 1983 and his medical degree from the same university in 1987. He was a resident at Denver General Hospital Affiliated Residency from 1988 to 1991 and was a fellow in the ACEP/EMF Teaching Fellowship program from 1993 to 1994 at the University of Texas-Southwestern Medical Center in Dallas.

He leaves behind his wife, Julia, and his two children, Conor and Maeve, as well as his parents, two brothers, and a sister. Memorial contributions may be made toward melanoma research in care of Dr. Sewa Legha, 2834 West NASA Blvd., Webster, TX 77598. ■

The Strategy of the Chest Pain Units (in Emergency Departments) in the War Against Heart Attacks: Proceedings from the First Maryland Chest Pain Center Research Conference

A Supplement to the *Maryland Medical Journal*

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INTERPRETIVE MEDICINE: A MANIFESTO

by Richard Horton, M.D., editor, *The Lancet*

In 1889, John Shaw Billings wrote that,
"Statistics are somewhat like old journals...most [of us] rarely use them, and find it troublesome to preserve them so as to have them easy of access; but when we do want them, we want them badly."

Much the same, I suspect, can be said about most of clinical medicine. We take it, like statistics and our journals, for granted. Indeed, the discipline of clinical medicine has been undergoing something of a crisis of confidence during the past quarter century or so. The rationale for maintaining or establishing discrete departments of medicine is unclear. Yet any serious discussion about our nervous anxiety towards the clinical art has been pushed to the margins of debate within academic medicine by the huge success of the specialties. The issue does not deserve such casual dismissal. Nevertheless, a resurrection of an old debate will be fruitless. A fresh approach to understanding the meaning of clinical medicine—as part of internal medicine and the specialties—is required. Interpretive medicine may provide one useful step forward.

It was Professor William Stanley Peart, professor of medicine at St. Mary's Hospital in London and anointed to his chair in 1956, who wrote, in 1970, that "There should be no more appointments to posts with the title of professor of medicine."¹ His argument was a familiar one, although it was sprinkled with the bitter taste of defeat. He admitted that,

"While fears of super-specialisation in university hospitals, whereby a man may know more and more about less and less, have to be recognised, it is useless to cling to the belief that specialisation can somehow be ignored. It is here, it is expanding, it will bring greater benefits to patients..."

He made an important distinction here. The general physicians would still survive outside of the university. But within the university, the professor of medicine deserved, according to Peart, to be shown the door.

Thirteen years later, Peart turned his argument around 180 degrees.² Under the title "Rebirth of the professor of medicine," he explained that his reversal rested "on the rapidity

of the changes in medicine brought about by new scientific thinking and modern technology.” He believed that,

“The role of a broadly based physician [is] even more important than before if the patient is not to suffer from the sheer weight of technology...”

The professor of medicine was, in his view, the “bridge” between laboratory science and clinical practice, giving “the lead amidst all the specialised divisions.” Surprisingly, a generation after these debates took place we are still discussing the place of general internal medicine in our practice. But, leaving aside the question about the future of internal medicine, is there a future for the clinical art itself?

The substance of Peart’s second argument seems right to me—academic clinical medicine does have a future, and it is bright. But that future depends on two, far from trivial, assumptions. One, that any programme for clinical medicine as a separate discipline must have a vision for practice and research to take it beyond the achievements of specialties. And two, that those within clinical medicine have the courage to do more than simply slice off parts of specialties that specialists are not themselves much interested in. Hypertension, diabetes, and stroke are obvious examples. And editors of general clinical journals should also have the courage not to accept for publication third-rate research that would be unpublishable elsewhere. Clinical medicine, in its welcome wish to be all things to all people, must resist the temptation to become the home for the homeless.

I would like to elaborate a little more, then, on three dimensions that I think could define the space of academic clinical medicine, which together constitute the beginnings of a manifesto for interpretive medicine.

First, there is the dimension of application. This is the bridge referred to by Peart. But the bridge is now much more between research and practice than between technology and practice. The huge growth in clinical research, and especially the importance of the randomised trial, has allowed a whole new set of problems to seep out. Most perplexingly, why is there a pervasive failure to translate the results of trial research into clinical practice?

The reasons—or excuses, depending on your point of view—for these failures are both methodological and interpretive. Methodological, because of failures in design, conduct, or analysis of a particular study. And interpretive, because of—most commonly—stubborn beliefs, overstretched inferences, and invisible generalisability.

The issue of generalisability is a special concern, I think. Take the debate over the safety of calcium channel blockers (CCBs), which has seen firm lines drawn between those who accept some validity in observational data derived from, for example, case-control study designs and those for whom nothing less than data from randomised trials will do.

When the Systolic Hypertension in Europe Trial group reported their important results,³ there was widespread acclaim that at last the safety of CCBs had been proven and that the unhelpful scare of the previous year or so could be forgotten. The Syst-Eur data are to be enthusiastically incorporated into influential U.S. guidelines on the management of hypertension, to be published soon.

But look at the detail: look, for instance, at the exclusion criteria for Syst-Eur. All patients were over 60 with isolated systolic hypertension. Patients were excluded if they had retinal

haemorrhages, papilloedema, heart failure, moderately raised serum creatinine concentrations, and a history of stroke, myocardial infarction, or any other severe cardiovascular disease. Syst-Eur was looking at an incredibly fit population, not at all those who were emerging from observational research studies as being at most risk.

Epidemiological science is the natural ally of the clinician. But when I look at most journals—general and specialty—I see very little evidence of this liaison.

Second, there is the dimension of synthesis, bringing knowledge from multiple disciplines and using information from multiple sources, in the patient consultation. Support for this argument is easily found.

The ASPIRE study—Action on Secondary Prevention through Intervention to Reduce Events—was published in *Heart* in 1996.⁴ It showed that recording of coronary risk factors in patient records was astonishingly incomplete. Among treatable risk factors, a quarter of patients with proven coronary disease remained hypertensive; over 75% had a high cholesterol; and only one in three patients was taking a β -blocker post-myocardial infarction. The scope for improvement seemed limitless.

This is the practice. But a research programme in interpretive medicine also exists to study the nature of clinical judgment and the influence of information sources, clinical experience, patient expectations and concerns, and the context of family, community, and culture in making these judgments. Our “knowledge” is often determined by cultural as much as data or theory driven forces, and we should be honest about that.

An example. The original meta-analysis about the safety of calcium channel blockers found dose-dependent relative risks of 1.3 to 3.0.⁵ These results were greeted with disbelief and several perfectly cogent arguments were constructed to explain away the findings. By contrast, the meta-analysis published in the *BMJ* on passive smoking⁶—with relative risks of cancer and heart disease of about 1.25 (and which were also based on observational data)—has been praised for its rigour, cheered for its message, and excitedly discussed by physicians, policy makers, and politicians, all of whom have vowed to act soon to eliminate smoke from our environment.

Reflect for a moment on how these systematic reviews have been received. Same type of data source, same method of combining data, similar risk estimates—but an entirely different reaction, not driven by either data or theory but affected by more obscure cultural forces, no less important for our understanding of the so-called evidence.

There is also a prevalent bias in our view of what research means. Edwin Bramwell, a professor of clinical medicine in Edinburgh, wrote in 1925⁷ about the “unfortunate use of the word research as the equivalent of laboratory methods,” which “belittle the importance of clinical observation and is undoubtedly responsible for the general impression that when the young graduate commences practice he relinquishes for good all opportunity of furthering the advance of medicine.” Bramwell agreed that “this misconception calls for correction.” And interpretive medicine is well placed to lead that corrective turn.

And finally, there is the dimension of reflection. By which I mean doing something that physicians hate to do and, if they must do it, feel decidedly uncomfortable when doing so. That is, stepping back a little from the bedside and thinking and writing about what we mean

by concepts such as disease, cause, truth, normal, health, mind and body, and even evidence.

This last dimension, which is essentially about inviting physicians to enter the territory currently occupied, rather uncertainly, by the philosophy of medicine, might seem self-indulgent and far removed from practice. But it seems to me even more important than ever. The dissonance that exists between public and profession is intensifying—complementary medicine, chronic fatigue syndrome, Gulf War illnesses, silicone breast implant-related health problems, priorities in international health, human rights issues—all of these have a central medical theme. But our largely skeptical and indifferent response to them has isolated physicians within an unattractive provincial and narrow island of medicine, a medicine that has become intellectually self-admiring of its scientific purity and, as a consequence, self-deluding and self-defeating.

Application, synthesis, and reflection—these are my personal wishes for a renaissance in clinical medicine. It is not concerned with hierarchies of evidence; it is not dependent on up-to-date literature alone as the arbiter of clinical decision making; it does not proselytise a bottom-line approach to the reading of new research. Rather, it is about preferring interpretations to conclusions, external validity to internal validity, context to the highly controlled—and artificial—experimental environment.

I think of these three axes as creating a three-dimensional space for interpretive medicine. It is one framework for the expression of clinical art.

In this supplement of the *Maryland Medical Journal*, the contributors under Dr. Raymond Bahr's charge have attempted to apply interpretive medicine to the heart attack problem in Maryland, USA, with the goal to challenge other states as well as other countries to do similarly. In this way the elements of application, synthesis, and reflection may indeed bring forth a renaissance in clinical cardiology.

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The early heart attack care strategy in the war against heart attack deaths utilizing the chest pain center approach in emergency departments

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ABSTRACT: *The focus of this symposium is Maryland's heart attack problem. The question is: Can the known evidence-based data on heart attacks be interpreted and applied in a systematic way that will unite efforts to reduce the significant heart attack deaths within the state? To determine this we need to go beyond what is currently being done and aim at a higher level of performance. Despite the medical advances in clot-dissolving therapies and minimally invasive surgeries, the acute prevention of heart attack death and damage has not been substantial. However, significant progress is possible. Better delivery systems, linking hospitals and communities, are needed. To accomplish this, emergency physicians and nurses, cardiologists, and paramedics need to form part of a team and have the support of the individual hospitals and the emergency medical system.*

Chest pain centers must function efficiently and cost effectively and bring about a community involvement that can significantly reduce heart attack deaths locally and, when combined with other hospitals, statewide.)

Hospitals need to be prepared. Hospital performance declines if studies are not being performed.¹ To prepare, hospitals must develop a learning curve similar to that which took place with the coronary care unit development (Figure 1). The beginning of this learning curve for the community and the chest pain centers was when thrombolytic therapy began. It allowed us to dissolve the clot quickly and save the patient's muscle and sometimes the vessel. Unfortunately, the coro-

Chest Pain Center Strategy

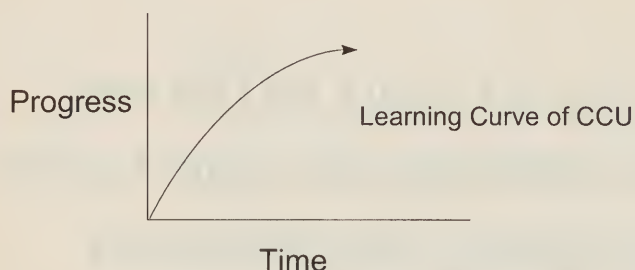


Figure 1. Chest Pain Center Strategy.

A need to recreate a learning curve similar to that seen with the coronary care unit movement.

nary care learning curve is diminishing and needs a booster to develop further early heart attack care. Chest pain centers have developed in response to this need. Most heart attack patients present with chest pain to the emergency departments throughout the nation. This is where we need to apply our strategy to enhance earlier care. This focusing is resulting in an exponential growth in chest pain centers throughout the United States. However, a facility cannot be called a chest pain center if it has only a protocol for patients crashing with acute myocardial infarction, showing only that it can reduce the time from emergency department arrival to thrombolytic therapy to less than 30 minutes.² This represents only 10% to 15% of patients presenting with chest pain in emergency departments. A systematic and comprehensive plan is needed for triaging and managing the other 85% to 90% of the patients.³

Recent studies have shown that this is possible; using the initial history and the electrocardiogram (EKG) patients can be stratified into one of five categories.⁴ These categories are called tracks or levels. Essentially, Track I and Track

II patients are those with acute myocardial infarction (with or without ST elevation) or worsening angina. Track V are patients with chest pain deemed not to be cardiac in origin. Track III and Track IV patients are at moderate to low probability for ischemic heart disease. These patients are usually placed in an observation area for six to twelve hours, where decisions can be made concerning their disposition. The best practice to date has been the use of cardiac markers and acute cardiac scan (Cardiolite) to stratify patients within a short period of time.³ This system allows patients in category IV to be safely discharged 80% of the time. After perfecting the Track IV protocol, the program can focus on expanding community outreach, thus allowing patients with soft chest symptoms (i.e., mild, intermittent, stuttering) to present to the hospital earlier (Figure 2).

In addition to the chest pain center functioning as a damage control area for heart attack patients, it also offers the opportunity to use the chest pain experience as a motivating force in changing behavior about other risk factor reductions needed by the patient. Thus disease prevention motivates health promotion and the two can be successfully linked together. In summary, the overall functions of the chest pain center include:

1. rule out myocardial infarction,
2. rule out myocardial ischemia,
3. rule out underlying coronary disease (stress testing), and
4. look for patients presenting with high risk factors

The chest pain center thus works as an encatchment area for ischemic disease patients within the community. The registry of such patients seen can go far beyond the expectations of just protecting the crashing (infarcting) patient. This concept reverses the direction of the United States Public Health Service, which is health promotion

and disease prevention, into disease prevention (damage control) and then health promotion (primary risk factor reduction). The chest pain experience in the chest pain emergency department helped motivate this change. Prevention takes on different forms: (1) vigilance in acutely preventing heart damage; (2) prudence in preventing coronary disease through life style changes; and (3) appreciation of what the heart can provide in our daily activities. Sometimes, it is through illness, and mistakes in dealing



- Expand the cardiac outreach program to reduce time delay outside the hospital for MI patients and most importantly, change the subset from MI patients to prodromal patients who have not yet had an MI.



- Perfect the "low probability for ischemic disease" protocol (Track IV).



- Move to a systematic and comprehensive approach to triaging and management of patients presenting with chest pain/chest discomfort syndrome.



- Focus initially on reducing the time to treatment of heart attack patients in the emergency department.

Figure 2.

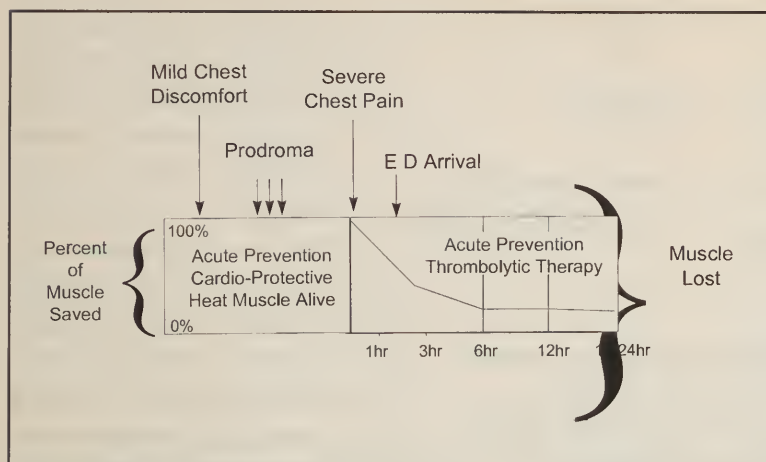


Figure 3. A New Paradigm in Early Heart Attack Care.

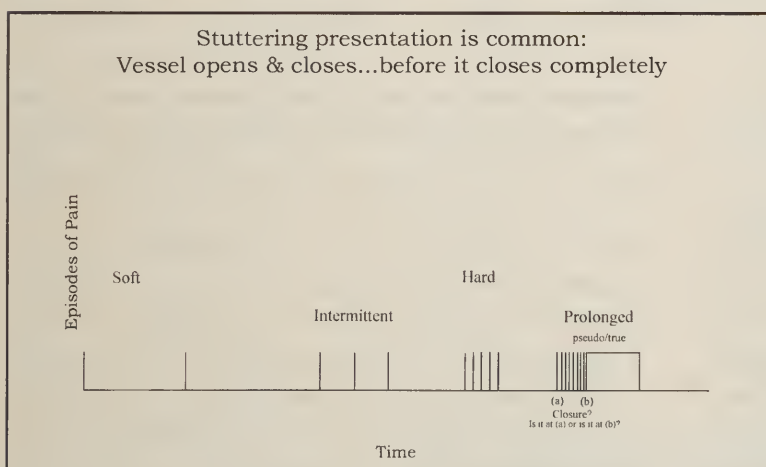


Figure 4. Chest Pain Symptoms.

with illness, that we learn to appreciate the value of this “heart-felt” action.

Patients do not have to crash (infarct) to get heart attack care. When the crash occurs, the outcome depends on many different and variable factors (e.g., Where does it

start? How close is the patient to the hospital? Can that hospital deliver timely thrombolytic therapy, or better yet, does it have a cardiac catheterization laboratory that is open and can immediately do angioplasty?).

To avoid the crash, we need patients to present at the earliest sign of symptoms. We know that prodromal symptoms occur in 50% of the patients presenting with heart attacks and that these symptoms occur over hours, days, and perhaps weeks.⁵ We must identify prodromal symptoms and take advantage of early action (Figure 3). This would significantly reduce heart attack deaths within the United States. The significance of prodromal symptoms of a heart attack have been in the medical literature for the last 75 years; only recently has there been emphasis on using the occurrence of these symptoms to fight heart attack deaths. We must detect prodromal symptoms early enough to prevent the myocardial infarction from occurring. We must determine if we can take a threatening myocardial infarction (MI), stabilize the patient, and prevent the need for thrombolytic therapy and primary angioplasty. In other words: abort the process.

In recent studies, where an aggressive early heart attack care community educational program aimed at prodromal symptom identification exists, patients appear to do much better and a higher percentage of MIs are aborted.⁶ Many of

these aborted MIs take place within the hospital after a stuttering presentation but before the prolonged and severe chest pain of coronary occlusion starts. Prompt thrombolytic therapy results in aborting the MI in a way

To create chest pain centers in EDs and focus attention on the heart attack problems:

- Recruit U.S. hospitals
- Mature these chest pain centers through guidelines, role model units, and studies from the chest pain groups.
- Research efforts to find the best critical pathways.
- Become cognizant of the environmental changes in this direction through the JCAHO (CQI), the HCFA Cooperative Cardiology effort, and the economics of managed care.

Figure 5. The Strategy of the Chest Pain Movement.

Formulate a strategy of chest pain awareness in the community

- Make chest pain an acute risk factor. Develop a learning curve for rapid expansion of our knowledge on chest discomfort and prodromal symptoms.
- Build a consensus support system.
- Develop a clear, simple message so that it can be widely and effectively disseminated.

Link the chest pain emergency department movement with the chest pain awareness educational program in the community for effective penetration at the local level in the 6,700 hospitals in the United

Figure 6. The Strategy of the Chest Pain Movement.

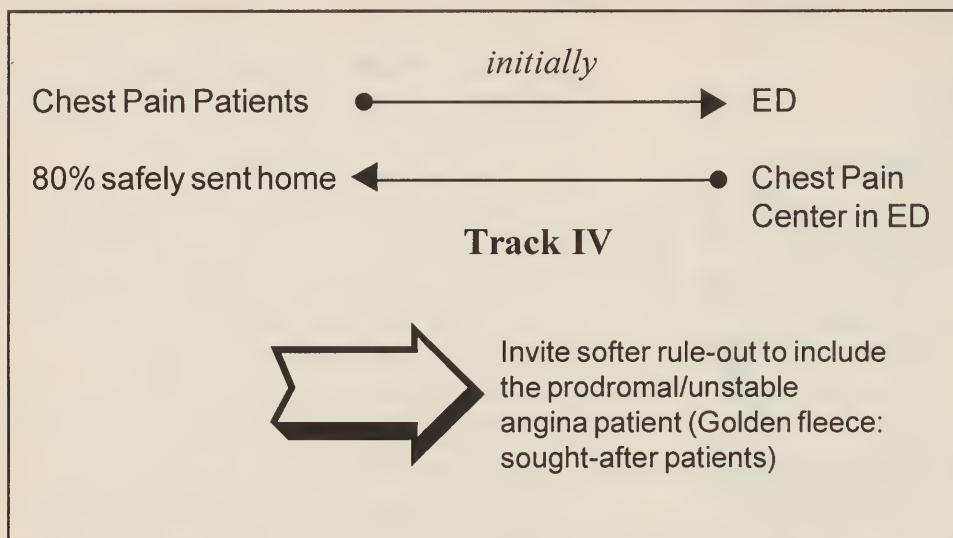


Figure 7. The Evolution of a Community "Sarcomere."

very similar to what was seen in patients treated within the first 70 minutes in the Myocardial Infarction Thrombolytic Intervention (MITI) trial.

Prodromal patients appear to exist as a mixed category. Not appreciating this fact may invalidate data from thrombolytic trials that measure "time to treatment within the first hour." Prodromal patients not only have stuttering presentations inside the hospital as well as outside the hospital, but they may also be more difficult in detecting the onset of occlusion even when the time from prolonged chest pain is measured (Figure 4). Serum myoglobin release precedes

the release of CK-MB by two to five hours; therefore, determining serum myoglobin may help in timing occlusion in prodromal patients. The concluding statement from Andreotti's article in the *New England Journal of Medicine* is relevant: "Occlusive coronary thrombosis in the early stages of myocardial infarction is a dynamic process, with repeated episodes of spontaneous reperfusion and occlusion occurring during a period of hours and perhaps days."⁷

The overall strategy of the chest pain center movement is to create chest pain centers in hospital emergency departments throughout the United States. This effort focuses on the heart attack problem and develops a learning curve of progress through research efforts, and offers best practice guidelines in a unique cooperation (Figures 5,6). The strategy is designed to rapidly take advantage of what is presently known in utilizing properly prepared community hospitals to serve as user-friendly check out points for patients identified in the community as having early manifestation of ischemic heart disease. The community effort can be

enhanced by educational programs that are based on prodromal recognition of early heart attack disease. This highlights chest pain as a risk factor and helps to unfold strategies needed to overcome seeming resistance points that prevent early treatment from taking place. The community "sarcomere" so developed will have for the "actin" an activated message within the community and for the "myosin" the muscle-saving properties employed within the chest pain center (Figure 7).

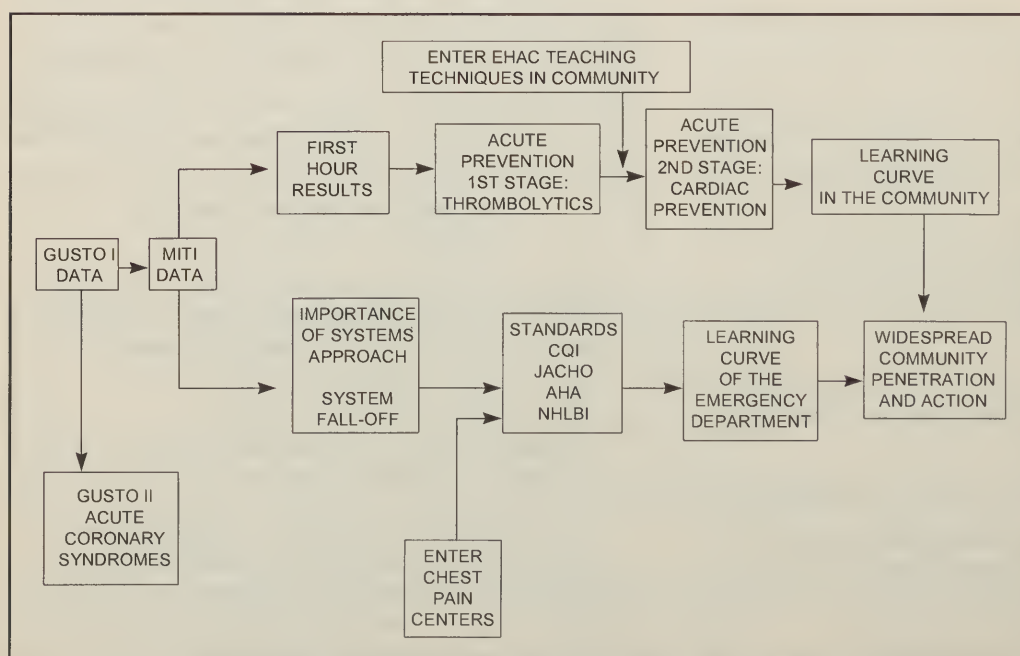


Figure 8. Overall EHAC educational and chest pain center strategy.

This symposium highlights risk stratification by improving history taking, providing up-to-date information on the use of cardiac markers, and highlighting the latest in what we perceive as the best way to risk-stratify patients in low to moderate risk ischemic categories, utilizing acute cardiac scanning. The hypothesis of the near- perfect chest pain center system will be tested, and whether Florida or Maryland has a better strategy to reduce significantly heart attack deaths within their respective states will be debated. This then becomes an effort to challenge ourselves and use this model as a prototype for other states to engineer similar type game plans and then compete to see who can do it better. This is what I believe Richard Horton, M.D., editor of the *Lancet*, meant with the term “interpretive medicine.”⁸ Our present knowledge allows us to interpret, engineer, and be creative with the chest pain center movement⁹ to take heart disease out of its position as the number one cause of death which it has occupied since the turn of the century (Figure 8).

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An EHAC Parable

In the beginning was the word, and the word was simple.
 Then the word expanded and became more intricate.
 And though many people heard the word, they could not remember it
 And others, who needed the word most, were not told.
 And so, they were afraid.
 Rare, then, was the one who could fulfill the word.

So the word was made simple. It was made clear.
 It was spoken to those who needed it most.
 It was spoken again and again,
 especially at the moment of greatest need.
 In time, those who heard the word remembered it, and acted.
 And lives were saved.
 And it was good.

—M.M. Newman

A clinician's approach to chest pain

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The proper approach to understanding a patient's complaint of chest pain has three essential components: 1) a careful history, elicited thoughtfully and without haste; 2) a targeted physical examination and a resting 12-lead electrocardiogram (ECG); and 3) a commitment to a "working diagnosis" that is limited to a) definite angina, b) probable angina, c) probably not angina, and d) definitely not angina. This entire process can usually be accomplished within ten minutes, and the same categorization can be made for acute myocardial infarction (AMI). Intentionally missing from this classification are the terms "possible angina" or "rule-out myocardial infarction (MI)." The latter two categories stifle critical thinking and serve as nothing more than intellectual wastebaskets. The previous four-way classification was used successfully in the Coronary Artery Surgery Study (CASS) more than 20 years ago and has stood the test of time. The CASS definitions are shown in **Table 1** and are derived from William Heberden's description presented to the Royal College of Physicians in London in 1772 (**Table 2**).

It is important to understand clinical characteristics that are not angina (**Table 3**) and the major non-cardiac causes of chest pain (**Table 4**).

Although the physical examination is generally thought to be of little help in distinguishing ischemic chest pain from other causes, it is far more helpful than physicians realize. Physical symptoms of this condition are presented in **Table 5**.

Specific approaches to patients judged to have definite or probable acute cardiac ischemia (ACI) — when the diagnosis is based on an unhurried, targeted history and physical examination — are determined largely by the accompanying 13-lead electrocardiogram (V_4r included). Regardless of the presence or absence of Q-waves, if ST elevations are present in two or more leads (i.e., elevations that reflect current level of injury, not ST elevations of pericarditis or those indicative of a left ventricular aneurysm), the patient should be admitted and triaged toward

TABLE 1. CASS definitions of angina
Circulation 1981; 63 (Suppl. 1:1-81)

- **Definite angina**
Substernal discomfort precipitated by exertion, with a typical radiation to the shoulder, jaw or inner aspect of the arm relieved by rest or nitroglycerin in less than ten minutes
- **Probable angina**
Has most of the features of definite angina but may not be entirely typical in some aspects
- **"Probably not" angina**
Defined as an atypical overall pattern of chest pain that does not fit the description of definite angina
- **"Definitely not" angina**
Question chest pain that is unrelated to activity, appears to be clearly of non-cardiac origin and is not relieved by nitroglycerin

reperfusion therapy, provided these findings are documented within 12 hours of symptom onset. The same would be true if the ECG showed left bundle branch block (LBBB) that was presumably new. There are two additional points that relate to the time factor: chest discomfort that persists for longer than 20 minutes frequently indicates that the ACI is actually an AMI; and the earlier that one is recording an ECG from the onset of symptoms, the less the likelihood the ECG will show definite changes of an AMI (i.e., if one is recording the ECG within the first hour of the onset of symptoms, it is less likely that Q-waves will be present or that ST changes will be apparent, and more likely that the ECG may even appear normal or indicate less objective changes such as heightened or peaked T-waves). The evolutionary changes of an AMI usually appear, however, within the first six hours of the onset of symptoms.

If ST elevations are not present, regardless of the presence or absence of Q-waves, the patient with definite or probable ACI should be admitted and not considered for reperfusion

TABLE 2. History

Definite angina

"A disorder of the breast marked with strong and peculiar symptoms . . . not extremely rare They who are afflicted with it are seized while they are walking (more especially if it be uphill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to continue; but the moment they stand still, all this uneasiness vanishes."

William Heberden to the Royal College of Physicians (1772)

TABLE 3. Features of "not angina"

Non-ischemic chest discomfort

- pleuritic pain: sharp or knife-like pain related to respiratory movements or cough
- primary or sole location: mid- or lower abdominal region
- any discomfort localized with one finger
- any discomfort reproduced by movement or palpitation
- constant pain lasting for days
- fleeting pains lasting a few seconds or less
- pain radiating into the lower extremities or above the mandible

Clinical practice guideline no. 10, unstable angina: diagnosis and management. AHCPR. Publication no. 94-0602, 1994.

unless subsequent ECG monitoring documents persistent ST elevation. If ST segment depressions are the sole abnormality in this setting, administration of fibrinolytic agents may actually be harmful, as the TIMI III-B study shows.¹

All of these patients should receive one aspirin (160 mg to 325 mg), nitroglycerin, and intravenous beta-blockers as they are admitted to the critical care unit. Serial measurements of serum markers for AMI (e.g., creatine kinase and its MB isoform, cardiac troponin T and I) can be expected to confirm the initial impression of AMI in patients with the designation of definite or probable AMI in 88% of cases; it was the same in the ISIS 2 trial, where the only entry requirement was that the patient be suspected of having an AMI.² At this stage of early decision making in the emergency department (ED), it is not critical that a distinction be made whether a patient is experiencing an AMI or one of the other ACI syndromes (i.e., unstable angina or a non-Q infarction). All such patients warrant admission to an intensive care or cardiac monitoring setting, and only those with requisite ST-segment elevations or LBBB should receive reperfusion therapy at this time.

Specific approaches to patients judged not to have ACI (probably not or definitely not) vary considerably, but it should be clearly stated that they do not warrant conventional admission to the hospital unless the alternate diagnosis is one of the serious conditions that mimic ACI (e.g., acute aortic dissection, pulmonary embolism, acute ulcer disease).

If the clinical paradigm of definite, probable, probably not, and definitely not is used correctly and takes into account the common variations of AMI presentation (such as more breathlessness and fatigue and less clear-cut chest pressure in the older patient, and awareness of the frequent location of referred cardiac pain, i.e., lower jaw, neck, interscapular region), unhelpful terms like "undifferentiated" or "atypical" chest pain can be avoided. Patients designated probably not or definitely not AMI should rarely "rule-in" ($\leq 2\%$ to 3%) in

TABLE 4. Major non-cardiac causes of chest pain
(exclude all pain above the mandible and below the umbilicus)

<u>Condition</u>	<u>Duration</u>	<u>Characteristics</u>
esophageal reflux	5 to 60 min.	visceral, recumbency, substernal, no radiation, relief with food, antacids
spasm	5 to 60 min.	visceral, spontaneous, substernal cold liquids, exercise relief with NTG
peptic ulcer	hours	visceral, burning, epigastric, relief with food, antacids, normal ECG
biliary disease	hours	visceral, epigastric interscapular colic, T-wave inversion
cervical disc	variable	superficial, positional, arm, neck
musculoskeletal	variable	superficial, positional, movement, local tenderness
hyperventilation	2 to 3 min.	visceral, substernal, tachypnea, anxious
thyroiditis	persistent	aggravated by swallowing, neck, throat tenderness

contrast to those patients designated definite or probable AMI who "rule-in" 88% to 90% of the time. In this context, the current use of the term "rule-out AMI" that is so pervasive in our medical jargon is totally unhelpful. It would make much more sense to use the terms "rule-in MI" for all patients considered to be the latter, and to limit the term "rule-out MI" for the former.

Patients designated probably not or definitely not also have a low pretest probability of any biological test being positive according to Bayes Theorem. Supporting this statement are the conclusions drawn by the coordinating committee of the National Heart Attack Alert Program after their careful evaluation of technologies for identifying ACI in the ED.³ Therefore, it seems unrealistic at the present time to expect any one of our existing methodologies to substantially refine the diagnostic accuracy that we can currently attain from this described clinical approach and the standard electrocardiogram. It also seems reasonable to recognize that there will be a comparatively small percentage of patients in whom the presence of serum markers for acute myocardial necrosis will be important to ascertain. These patients would seem ideally suited for referral to some "observational area" within the hospital setting for continued observation for a period of time less than 18 to 24 hours. Such an approach will only be obviated when the "ideal" cardiac serum marker (containing a 100% sensitivity and 100% specificity within one hour of the onset of symptoms) is identified that will remain as sensitive and specific for the next 24 to 48 hours.

Exactly what role any form of stress testing plays in the probably not and definitely not patient populations is less than clear. It is widely appreciated that the sensitivity and specificity of conventional exercise electrocardiographic testing is improved by the addition of nuclear imaging techniques and can reach as high as 90% sensitivity in patient populations known to have coronary artery disease. However, in these specific populations the incidence of coronary artery disease falls well below 40%, and according to the Bayesian principles, the predictive accuracy of such testing falls well below acceptable levels.⁴

A positive test, for example, has a high probability of being a false positive. The addition of echocardiographic imaging to stress testing (either exercise or pharmacologic) held the promise of a solution to this problem by revealing new regional wall motion abnormalities or by demonstrating the failure of myocardium to further recruit with infusions of Dobutamine. As this technique is applied to broader populations, however, it is apparent that segmental wall motion abnormalities are not specific for coronary artery disease and can be demonstrated, sometimes quite dramatically, in patients with cardiomyopathy and hypertensive heart disease. Until ongoing investigative studies show better results with all forms of imaging (including fast-CAT scanning and magnetic resonance angiography), we are left to concede that coronary angiography remains the only way of truly assessing the presence or absence of coronary artery disease. Perhaps efforts to develop a bedside method to obtain satisfactory coronary arteriograms makes more

TABLE 5. Features of ischemic type chest discomfort

Physical exam

**Usually of little help in making diagnosis of coronary artery disease*

1. diminished regional pulses
2. vascular bruits
3. evidence of aortic abdominal aneurysm
4. soft systolic murmur
5. paradoxical splitting of Sw

**Of critical importance in AMI*

sense than was initially thought. The problem, however, is that the demonstration of a coronary lesion by angiography does not necessarily indicate its functional significance.

These comments should not be interpreted as an indictment against the use of stress testing when it is truly uncertain whether or not the patient has coronary disease; instead, it is a call for reassessment of the perceived value of such testing. It certainly questions whether or not we should be spending three billion dollars annually to assess whether a patient has "probably not" or "definitely not" angina. A clinical paradigm using the "definite," "probably yes," and "probably no" approach does far better than physicians have given credit for in this era of reliance on technology. At a minimum, technology assessment should be reserved for those patients assigned to the "probably not" category — and whose condition is truly uncertain.

The call for further development of chest pain clinics makes sense only if they are to be used, as has been proposed by Raymond Bahr, M.D., and his colleagues, to capture persons who are having prodromal symptoms prior to an ischemic event rather than using them to manage persons who have already had an acute ischemic event.

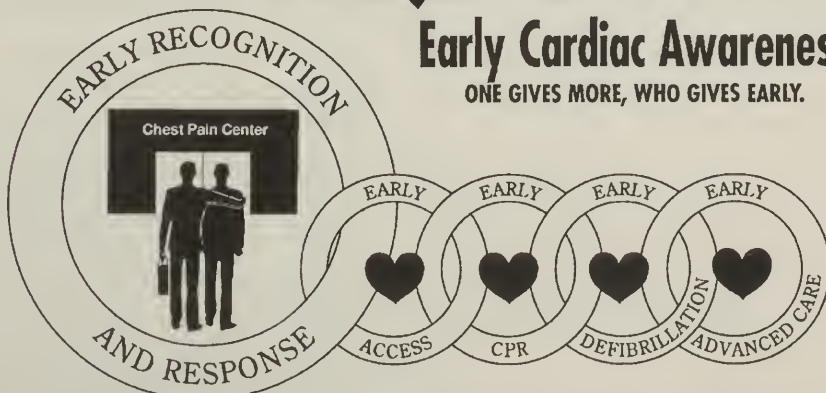
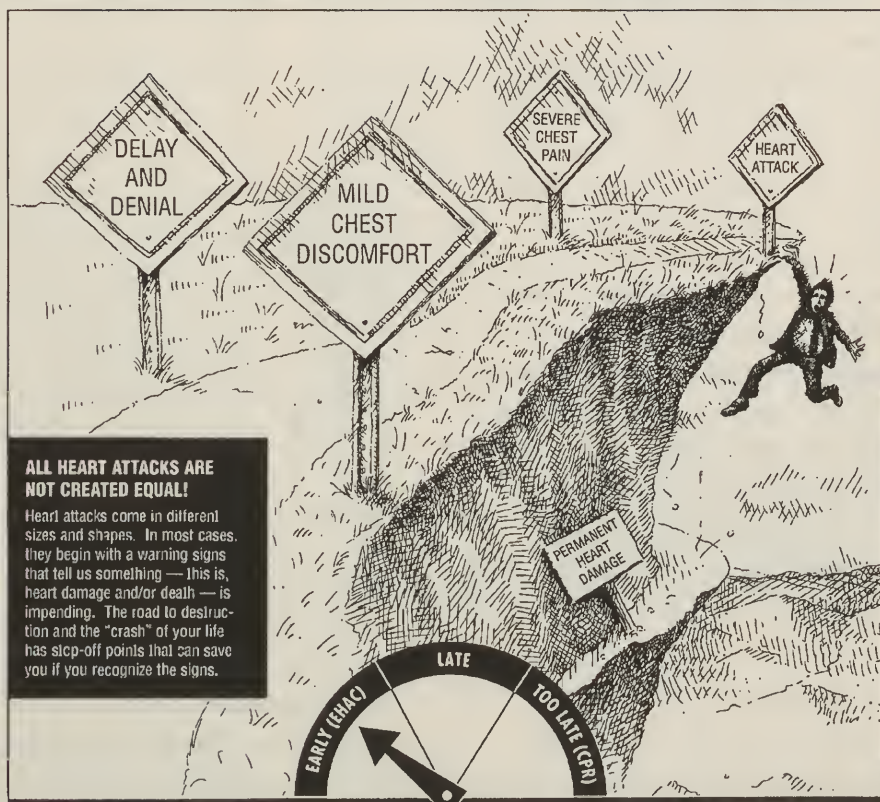
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Cardiac markers in the assessment of acute coronary syndromes

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ABSTRACT: Biochemical markers provide clinicians with an important tool for the assessment of acute coronary syndromes. Biochemical markers, including total creatine kinase (total CK), creatine kinase-MB (CK-MB), the MB isoforms, and myoglobin, as well as the troponins — cardiac troponin T (cTnT) and cardiac troponin I (cTnI) — are all used for assessment of the suspected acute myocardial infarction (AMI) patient. In the context of myocardial infarction (MI) diagnosis, total CK is a relatively sensitive marker, but it lacks myocardial specificity because skeletal muscle contains high concentrations of CK. CK-MB is the benchmark for biochemical markers and has both high sensitivity and specificity; however, CK-MB is also present in skeletal muscle and is not diagnostic until eight to twelve hours after onset of symptoms. The MB isoforms are diagnostic earlier but have the same cardiac specificity issues as CK-MB. Myoglobin becomes abnormal about one hour after onset of symptoms and is a sensitive marker for MI; however, myoglobin is cleared quickly and is not cardiac specific. Both cTnT and cTnI are cardiac specific and show high sensitivity and specificity for MI. Risk stratification of acute coronary syndrome patients is another role for biochemical markers; CK-MB, cTnT and cTnI have all been proposed for this function. Compared with CK-MB, both cTnT and cTnI are better able to predict short-term mortality following the index event. Analysis using a logistic regression model that included the electrocardiogram, cTnT, and cTnI showed that cTnT was the most useful marker for risk stratification. Finally, cTnT was reported to be able to predict which patients will benefit from

Clinical symptoms, the electrocardiogram (ECG), and biochemical markers of myocardial injury are all essential data for global assessment and development of treatment decisions for patients presenting with “acute coronary syndromes,” a term that describes the continuum of cardiac ischemia and myocardial infarction (MI) ranging from:

Angina \Rightarrow Unstable Angina \Rightarrow non-Q-Wave MI \Rightarrow Q-Wave MI

Careful assessment of clinical symptoms is obviously of paramount importance. However, symptoms can be highly variable, particularly in elderly patients. The ECG is a most important tool and should be performed soon after arrival of the suspected MI patient to the emergency department.¹ Although the ECG is diagnostic in only 24% to 60% of patients on presentation,² patients having either ST segment elevation $>1\text{mV}$ in contiguous leads and new left bundle branch block are candidates for immediate reperfusion therapy.¹ Biochemical markers have been considered the “gold standard” for the diagnosis of MI³ and are a particularly important part of assessment of patients presenting with non-diagnostic ECG. Care of the non-diagnostic ECG patient has been facilitated by establishment of specific treatment areas intended for the systematic and cost-effective treatment of patients suspected of having acute MI; these areas are often termed chest pain evaluation centers.⁴

Biochemical marker testing is performed for diagnosis of acute MI, risk stratification, assessment of reperfusion status after thrombolytic therapy, assessment of re-infarction or extension, and estimating the amount of infarcted tissue (infarct sizing). Of these indications, utilization for diagnosis of acute MI and risk stratification in the non-diagnostic ECG patient are most important and relevant applications. Although there are views both pro⁵ and con⁶ concerning the need for rapid availability of biochemical markers, recent literature showed an association with both reduction in length of hospitalization and overall laboratory costs in institutions producing shorter turnaround times.⁷ Biochemical markers with real-time potential for utilization in risk stratification and diagnosis of acute MI will be the focus of this review.

The ideal marker of myocardial injury

A key characteristic of the ideal biochemical marker is rapid release after myocardial injury to levels above the normal reference interval or “cutoff”; the marker should remain elevated for several hours to several days. The marker should

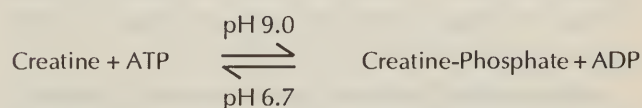
show a predictable release profile, with an initial rise, and then a fall pattern when the acute event is resolving; variations in this rise and fall pattern should indicate complications, such as extension or reinfarction. The ideal marker should be low or near-zero in the absence of disease, but have a high tissue/plasma ratio, such that even minor myocardial injury can be detected sensitively. Also, the total amount of marker released over time should be proportional with the extent of myocardial injury. The marker must have high specificity for myocardial tissue.

Measurement of the ideal marker would be rapid; that is, results are available within a few minutes, and require a small volume of whole blood in a device that would minimize risk of exposure to infectious agents. The assay would be “site neutral,” so that testing could be performed at any location inside or outside an institution. The ideal test would be inexpensive, and the results would yield reproducible results that are standardized to match data from other testing locations and from other institutions.

Results of the ideal biochemical marker would be quantitative, to allow for a continuum of interpretations corresponding to the spectrum of acute coronary syndromes. There should be a strong association between the marker and both short- and long-term risk stratification, giving caregivers a clinical tool to accurately identify and intervene in cases at high risk of adverse outcome.

Biochemical markers

Total Creatine Kinase (Total CK). Creatine kinase (CK) is a cytosolic enzyme that catalyzes the phosphorylation of creatine as shown in the following reaction:



Creatine-phosphate may be considered an energy “warehouse” and CK the means for both storing and recalling this energy. At relatively high intracellular pH, when energy demands are low, CK facilitates storage of high-energy phosphate bonds on creatine (forward reaction). As cellular energy demands increase, there is production of lactic and other acids which lowers pH. This causes CK to catalyze re-formation of the high-energy phosphate bond for rapid use (reverse reaction). Presumably because of this role in energy utilization, CK is found in high concentrations in striated muscle and numerous other tissues.⁸

Structurally, CK is a dimeric enzyme composed of M and/or B subunits that associate to form CK-MM (CK-3), CK-MB (CK-2), and CK-BB (CK-1) isoenzymes. “Total CK”

TABLE 1. Characteristics of various biochemical markers of myocardial injury.

Biochemical Marker	Molecular Weight (daltons or g/mole)	Cardiac Specific?	Renal Function dependent?	First Rise	Duration of Elevation
Myoglobin	18,000	No	Yes	1-3 hours	12-24 hours
Total Creatine Kinase	85,000	No	No	4-8 hours	36-48 hours
CK-MB, mass assays	85,000	++	Yes	3-4 hours	24-36 hours
MB Subtypes (MB ₂ / MB ₁ Ratio)	85,000	++	No	2-4 hours	16-24 hours
Cardiac Troponin T	37,000	++++	Yes	3-4 hours	10-14 days
Cardiac Troponin I	23,500	++++	Yes	4-6 hours	4-7 days

concentration refers to the cumulative activity of the MM, MB, and BB isoenzymes in patient samples.

CK-MM is the predominant CK isoenzyme in both skeletal and myocardial striated muscle. These tissues differ, however, in that CK-MB composes approximately 20% to 30% of the total CK in myocardial muscle.⁸ In skeletal muscle, CK-MB composes only 0% to 3% of total CK; CK-BB predominates in brain and other tissues.⁸

As indicated in **Table 1**, total CK generally achieves abnormal concentrations in circulation approximately four to eight hours after the onset of clinical symptoms in MI patients. Although total CK is generally considered a diagnostically sensitive marker of myocardial injury, up to 2% of MI patients show "intranormal" elevations, so values must be interpreted carefully.⁹ Intranormal elevations occur because total CK is present in circulation due to normal tissue turnover and the normal reference interval shows substantial interindividual variation based on muscle mass, age, sex, or race.⁹ As indicated in **Table 1**, total CK is not myocardial specific; however, fractionation of the CK-MB isoenzyme can greatly improve cardiac specificity.

Creatine kinase-MB (CK-MB). CK-MB must be considered the benchmark for biochemical markers of myocardial injury and, as such, is the basis for assessment of other markers. Although CK-MB release is highly specific for myocardial injury, skeletal muscle may contain up to 3% CK-MB and has higher total CK activity per gram of tissue.⁸ This potentiates a specificity problem in patients with concomitant myocardial and skeletal muscle injury.

Measurement of CK-MB may be accomplished by either "activity" assays, which assess the functional ability of CK-MB for converting substrate to product, or "mass" assays (or immunoassays), utilize specific antibodies for recognition of CK-MB as a protein. Electrophoresis is an example of an activity assay because the concentration of CK-MB in the patient sample is reflected by the amount of product produced in the assay. Mass assays must be considered the "state of

the art" for CK-MB measurement because they characteristically have better analytical sensitivity and better precision than activity assays.⁹

In an attempt to confer more cardiac specificity to CK-MB, a "CK-MB Relative Index" is frequently calculated according to the following equation:

$$\text{CK-MB Relative Index} = 100\% (\text{CK-MB} / \text{total CK})$$

For mass assays, CK-MB Relative Index values exceeding 2.5% are associated with a myocardial source of CK-MB.¹⁰ For activity assays, the CK-MB Relative Index varies but is usually in the range of 5%.

The characteristic rise and fall of CK-MB in serial measurements is nearly pathognomonic for diagnosing MI.⁹ Utilizing "mass" CK-MB assays in a strategy that included sampling at presentation, 3 hours, 6 hours, and 9 hours, Gibler et al. documented a sensitivity of 100% and specificity of 98.3% for MI diagnosis in nondiagnostic ECG chest pain patients.¹¹ Despite this excellent performance, CK-MB is not the ideal marker because it requires eight to twelve hours after symptoms for use in diagnosis,^{9,11} and tissue specificity is an issue, as discussed above.

Isoforms or subtypes of CK-MM and CK-MB. The CK-MM and CK-MB isoenzymes can each be fractionated into subtypes, or "isoforms."¹² CK-MM consists of three subtypes, termed MM₁, MM₂, and MM₃ based on their relative migration toward the anode on high-voltage electrophoresis. Of the three CK-MM isoforms, MM₃ accounts for over 95% of the CK-MM activity within tissue. Upon release into circulation, the MM₃ tissue isoform is irreversibly converted to MM₂, and then MM₁ plasma forms by successive enzymatic cleavage of the carboxy-terminal lysine of first one M subunit, and then the other.¹³ The MM isoforms are virtually never utilized clinically because CK-MM is not myocardial specific.

Although the number of the MB subforms existing in nature remains controversial,^{12,14} two protein bands are con-

sistently identified after high-voltage electrophoresis. These proteins, designated MB₂ and MB₁, are similar to the MM isoforms in that only the cathodal MB₂ isoform is present within tissue.¹³ MB₂ is converted to MB₁ by irreversible enzymatic cleavage of the carboxy-terminal lysine of CK-MB's only M subunit.¹³ Normally, the concentration of MB₂ and MB₁ are equal;¹⁵ with tissue injury, there is release of MB₂ and an increase in the ratio of MB₂/MB₁ within the first few hours. As indicated in Table 1, the MB isoforms have many similar characteristics to CK-MB.

The MB isoforms have been used in clinical applications, either by temporal monitoring of MB₂ concentrations or by formulating an MB₂/MB₁ ratio.^{12,14} The MB₂/MB₁ ratio has demonstrated promise for use in the diagnosis of MI within the first few hours after onset of symptoms.^{15,16} The MB₂/MB₁ ratio becomes abnormal before CK-MB in patients with MI, with high diagnostic sensitivity and specificity.¹⁶ The negative predictive value of the MB₂/MB₁ ratio is also reportedly high, leading to suggestions that the MB isoforms may be useful for eliminating unnecessary admissions to critical care unit beds and a substantial reduction in the associated costs.¹⁶ These MB isoform data are promising, but must be considered preliminary.

Myoglobin. As indicated in Table 1, myoglobin is a relatively small protein that is abundant in the cytosol of striated muscle cells, both skeletal and myocardial. Myoglobin is released rapidly after tissue injury and may be elevated as early as 1 hour after myocardial injury.¹⁷ Myoglobin is also cleared rapidly by renal excretion, so abnormal levels may return to baseline values in six to twelve hours.¹⁷

Several authors have reported that serial myoglobin measurement, compared to CK-MB,^{18,19} represents a more diagnostically sensitive marker of myocardial necrosis in the first three to four hours following presentation. Myoglobin, however, is not a cardiac specific marker because the form released from both skeletal and myocardial tissue is identical. Therefore, myoglobin may be elevated in conditions such as skeletal muscle trauma, intramuscular injections, extreme exercise, and other conditions, in addition to MI.¹⁷

As with any clinical test, the diagnostic specificity reported for myoglobin is highly dependent on the population examined. The diagnostic sensitivity of myoglobin for detecting myocardial tissue injury indicates that the negative predictive value is very high and may have clinical use.^{18,19} Although the rapid clearance of myoglobin is a caveat, if myoglobin values do not rise within three to four hours after presentation with acute symptoms, then the likelihood of MI is low. Prudent utilization of myoglobin, however, is as a component of a

cardiac panel, along with a later marker so that events occurring over six to twelve hours in the past can be detected with confidence.

Proteins of the troponin complex. The contractile apparatus of skeletal and myocardial striated muscle is composed of myosin-containing thick filaments, surrounded by an octagonal array of actin-containing thin filaments. The thin filament includes an actin helix bordered by tropomyosin strands that periodically contain a three-member "troponin complex." The sliding filament model of muscle contraction proposes that an ATP-dependent actin and myosin interaction is "triggered" by release of calcium ions during electrical depolarization. Released calcium ions bind to the troponin complex—consisting of troponin I, troponin T, and troponin C—to change its conformation and result in contraction.

Key characteristics of troponin T and troponin I are listed in Table 1. Functionally, troponin T serves to bind the troponin complex to the tropomyosin strand of the actin thin filament. Troponin I functions to inhibit the activity of actinomycin ATPase. Troponin C serves to bind four calcium ions and regulates contraction. Great interest has been generated by the proteins of the troponin complex because cardiac-specific isoforms of troponin T and troponin I have been purified, allowing antibody production and development of cardiac-specific immunoassays.²⁰⁻²² The amino acid sequence for troponin C from cardiac and skeletal muscle tissue is identical, precluding use as a marker.

Cardiac troponin T (cTnT) and cardiac troponin I (cTnI) represent a new generation of biochemical markers that may provide an additional clinical tool for assessment of the acute coronary syndromes. Although cTnT and cTnI are both structural proteins, and therefore insoluble, each has a hypothetical "cytosolic" pool that is released into circulation after cell injury. The cytosolic pool for cTnT has been reported at 6%²³ to 8%,²⁴ while the soluble cTnI pool is reportedly 2.8%.²² Therefore, cTnT and cTnI must be considered two different proteins because they differ in biological function, molecular weight, cytosolic pool, and other important characteristics that may impact on their clinical utilization.

Cardiac troponin T (cTnT). Currently, there is one assay for cTnT that is cleared by the Food and Drug Administration (FDA) for clinical use (from Boehringer Mannheim Corp., Indianapolis, IN). Cardiac troponin T measurements with this assay have been compared to the CK-MB mass standard for use in the diagnosis of MI.²¹ Wu et al. found that cTnT was a diagnostically sensitive marker in the first six hours after onset of symptoms and remained so for the several days thereafter,²⁵ presumably because of the prolonged release of the structurally bound cTnT pool. Although cTnT has demonstrated high sensitivity, this study found that cTnT's

diagnostic specificity was lower than the CK-MB benchmark. This finding contrasted with results from some studies,²³ possibly because a substantial number of patients in this study population had minor myocardial damage due to unstable angina, lowering diagnostic specificity.²⁵

Cardiac troponin T increases have been documented both in patients with end-stage renal disease²⁶⁻²⁸ and with skeletal muscle injury.²⁹ It is noteworthy that a more cardiospecific modification of the cTnT assay used in these studies is now available and may improve performance further.³⁰ Also, cTnT elevations associated with renal failure and skeletal muscle injury may be a harbinger of an adverse outcome, so knowledge of the true significance must await completion of appropriate outcome studies.

Cardiac troponin I (cTnI). Currently, three quantitative immunoassays are cleared by the FDA for clinical measurement of cTnI: one from Sanofi-Pasteur, Chaska, MN, and two from Dade-Behring, Glasgow, DE. One qualitative cTnI test, intended for point-of-care use, has also cleared the FDA. All have demonstrated substantial equivalence with CK-MB mass assays and excellent potential for clinical use in the diagnosis of myocardial infarction.³¹⁻³³ Therefore, cTnI may have an important role in real-time strategies for evaluating acute coronary syndrome patients, an area that has been of intense interest, discussion, and study over recent years.⁵ Data are rather limited, but they indicate that cTnI is a specific marker in cases involving skeletal muscle injury and renal failure.^{33,34}

Risk stratification

Over the past few years, careful review and meta-analysis of a number of clinical outcome-based studies have demonstrated that patients with acute cardiac ischemia, in whom cTnT was elevated, were at increased risk for adverse events, including myocardial infarction and/or cardiac death.^{35,36} However, CK-MB increases, even below the reference interval, have also been recognized as being associated with an adverse outcome.³⁷

A study designed to compare the usefulness of CK-MB, measured by a state-of-the-art mass assay, cTnT, and the ECG for risk assessment, was performed as a substudy of the GUSTO IIa trial.³⁸ This GUSTO IIa substudy included 854 patients, all of whom had symptoms of cardiac ischemia within 12 hours of enrollment and an abnormal ECG. GUSTO IIa showed that the higher the cTnT level at presentation, the greater the risk of 30-day mortality.³⁸ Also, patients who

TABLE 2. Cardiac troponin T levels and outcomes at 150 days from the FRISC study.³⁹

Cardiac Troponin T Concentration, ng/mL	Cardiac Death	Cardiac Death or Myocardial Infarction
<0.06	0%	4.3%
0.06-0.18	2%	10.5%
0.18-0.62	2%	16.0%
0.62-2.12	7%	20.0%
≥2.12	9%	17.0%

tested positive for cTnT had a three-fold increase in morbidity compared with patients who tested negative. Further, cTnT was the most powerful predictor of 30-day mortality.³⁸ Combined analysis, in a model that included the ECG, cTnT, and CK-MB, confirmed that: 1) cTnT added the most information regarding risk of 30-day mortality, and 2) CK-MB provided no added value beyond that provided by the ECG and cTnT.³⁸

In a separate trial termed the FRISC study, peak cTnT concentration occurring in the 24 hours after presentation was correlated with 150-day cardiac death and/or MI in 976 patients, all of whom presented with unstable coronary artery disease.³⁹ A key finding of the FRISC study was that risk of an adverse cardiac outcome increased as the cTnT value increased, as indicated in **Table 2**. This study concluded that cTnT measurement in the first 24 hours provided valuable prognostic information over the following five months that was independent of age, hypertension, number of antianginal drugs, and ECG changes.³⁹

A separate aspect of the FRISC study investigated if cTnT concentrations could be useful for identifying patients with unstable coronary artery disease who might benefit from therapeutic intervention. This issue was investigated by measuring peak cTnT levels in 971 patients who received either placebo or low molecular weight heparin in short-term (six-day) or long-term (five-week) regimens.⁴⁰ Among patients having cTnT levels <0.1 ng/mL at initial presentation, short-term treatment reduced the incidence of death and/or MI from 2.4% to 0% compared to placebo ($p = \text{not significant}$). In patients with cTnT ≥0.1 ng/mL, the incidence of death and/or MI was reduced significantly from 6.0% to 2.5% in the placebo versus short-term treatment groups, respectively ($p < 0.05$). With long-term low molecular weight heparin treatment, patients having cTnT ≥0.1 ng/mL had outcomes of death and/or MI at a rate only one-half that of the placebo group (7.4% vs. 14.2%; $p < 0.01$). Further, cTnT levels <0.1 ng/mL identified a low-risk group in whom death and MI showed no difference between the treated and placebo groups. The authors concluded that elevated cTnT concentrations clearly

TABLE 3 . Relative value of the serum markers cardiac troponin T (cTnT), cardiac troponin I (cTnI), and the electrocardiogram (ECG) in the prediction of 30-day mortality.

Model	Variable	χ^2	p-value
Univariable	cTnT	21.0	<0.001
Univariable	ECG	14.2	0.003
Univariable	cTnI	12.3	0.002
Model	Added Variable	Added χ^2	p-value
cTnI, ECG	cTnT	8.03	0.045
cTnT, cTnI	ECG	9.96	0.019
cTnT, ECG	cTnI	0.84	0.675

identified patients who would benefit from long-term treatment with low molecular weight heparin.⁴⁰

Cardiac troponin I has also been investigated for use in risk stratification of acute coronary syndrome patients and compared with CK-MB mass values.⁴¹ One study of 1404 unstable angina or non-Q-wave MI patients used an endpoint of 42-day mortality. Cardiac troponin I of ≥ 0.4 ng/mL appeared to indicate increased risk of mortality (risk ratio = 3.1), even in patients whose CK-MB measurements were not abnormally elevated. This study concluded that cTnI provides for the early identification of patients at increased risk of death.⁴¹

Because both cTnT and cTnI have been used for risk stratification, the performance of these markers was compared, using 30-day mortality as the outcome, in 770 patients⁴² as an extension of GUSTO IIa. Results of this study showed that the area under the Receiver Operator Characteristic (ROC) curve for cTnT was larger, at 0.69, than the value for cTnI, at 0.64. Because the area of the ROC curve relates directly to test performance and is independent of cutoff, cTnT was better able to predict 30-day mortality than cTnI.

To examine the relative significance of the troponins and the ECG for predicting 30-day mortality, a logistic regression model that examined cTnT, cTnI, and ECG as variables, either alone or in combination, was used. **Table 3** summarizes the results. Considering each variable individually, cTnT was most useful for predicting 30-day mortality. Further, when cTnI and the ECG were put in the logistic regression model first, cTnT added significant information ($\chi^2=8.03$, $p=0.045$). However, when cTnI was the variable added to a cTnT and ECG model, there was no significant increase in the ability to predict 30-day mortality. Thus, cTnT provided the most information regarding prediction of 30-day mortality in the GUSTO IIa population.⁴²

In all comparisons of cTnI and cTnT for risk stratification and acute MI diagnosis, it is important to note that the charac-

teristics of the marker may be dependent on the assay used for measurement, as well as on the patient population examined.

Conclusion

The evolution of new markers, such as the troponins, and improved assays for markers, such as myoglobin, CK-MB, and the MB isoforms, have provided clinicians with new tools for assessing patients within the spectrum of acute coronary syndromes. The utilization of these markers for assessment strategies in the suspected MI patient are established in part. The increased realization that cTnT and cTnI have prognostic value, and that biochemical markers may have a role in guiding therapy,

has the potential of altering the way that caregivers view the acute coronary syndromes. It is unknown whether the increased cTnT, cTnI, and CK-MB concentrations observed in unstable angina patients represent actual myocardial necrosis or high-risk coronary artery disease. It is apparent, however, that outcomes among unstable angina patients having increased marker values are similar to patients "ruled in" for MI. Although this is an exciting era, much work remains in the area of biochemical markers of the acute coronary syndromes. For example, substantial challenges remain for the improved assessment of patients with prodromal symptoms. Also, discovery of earlier myocardial specific markers, as well as continued development of site-neutral and more sensitive assays for existing markers, is clearly needed.

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Cost effective nuclear scanning in a comprehensive and systematic approach to the evaluation of chest pain in the emergency department

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ABSTRACT: *The cost of evaluation and treatment of patients presenting to the emergency department with chest pain is estimated in billions of dollars. Current standards of practice, however, cannot reliably distinguish between high- and low-risk patients. Efficient management of this population requires that we reduce: 1) delays in therapy, 2) soft admissions, 3) inappropriate dispositions, and 4) cost. In a multidisciplinary effort, the acute cardiac team developed a comprehensive evaluation and triage strategy based on risk. Our data suggest that we have been able to accomplish all four goals, including a reduction in overall costs through the use of perfusion imaging.*

Chest pain is an especially enigmatic problem for the emergency department (ED) physician. Most patients presenting with chest pain have nonacute problems and do not require hospital admission, but chest pain is also a common manifestation of far more serious, high-risk conditions.¹ These include some relatively rare conditions such as aortic dissection, pulmonary embolus, and pericarditis, as well as acute myocardial infarction (MI), which is a major concern. Even experienced ED physicians may have difficulty distinguishing high-risk ischemic and/or infarction presentations from those retrospectively labeled nonacute conditions.²⁻⁴ With the relative insensitivity of the initial electrocardiogram (ECG),⁵ especially for the posterior and lateral MI, it is probably remarkable that the missed MI rate, reported as 4% to 6%,⁶⁻¹¹ is not higher. Yet, this fact necessitates a liberal admission policy which, by some estimates, leads to the admission of up to 50% of patients who are subsequently discharged with a nonischemic diagnosis.¹² This must be balanced, however, against the fact that patients with acute MI and a nondiagnostic ECG—while at relatively low risk for significant morbidity/mortality if admitted—are at high risk if discharged.^{13,14}

Inevitably, this tenuous balance of risk and cost is further threatened by the changing health care environment. Two opposing forces have had a significant impact, upgrading a problematic situation to a true dilemma. The introduction of thrombolytics has provided a mechanism for effective therapy for acute MI when applied in a timely manner. However, health care reform has introduced an opposing cost containment and cost-effectiveness element. The availability of an effective yet time-sensitive intervention produces the need for earlier identification of patients at risk in the environment. This need increases the pressure on imperfect initial evaluation tools.⁵ Logically, under the existing system, this would produce an increase in the number of admissions, most likely among patients without true ischemic events. At the other extreme, health care reform and its increasing application of capitation demands a careful examination of costs and cost-effectiveness. These competing forces have prompted a search for solutions that will enable health care to meet four competing goals: 1) to reduce delays to therapy and disposition, 2) to reduce the frequency of soft admissions, 3) to reduce the probability of inappropriate disposition, and 4) to reduce cost while increasing efficiency.

One solution appeared to be the dedicated chest pain unit or chest pain emergency room. Dedicated chest pain units were not initially developed for cost-effectiveness, but rather for improvement in the quality of care for chest pain patients.^{9,15} Their development was based on the theory that specialized professionals and services would provide more effective and appropriate therapy for the evaluation of the chest pain patient. Although no study has demonstrated global cost savings, several studies among the premier chest pain units in the country have demonstrated an improvement in care, as well as an overall reduction in cost of patient evaluation.¹⁵⁻¹⁷ This cost reduction is partly due to the implementation of a systematic strategy, which appears to improve efficiency regardless of the system itself.¹⁸ However, early risk stratification and appropriate triage of the chest pain patient remain fundamental problems with the available initial triage diagnostic tools.

In October 1993, a group of health care workers at Virginia Commonwealth University, Medical College of Virginia Hospital formed a multispecialty team (the Acute Cardiac Team, or ACT) to develop a systematic strategy to meet these four goals. The team quickly determined that the initial strategy would consist of a comprehensive triage system, that would lead to a series of five critical pathways (levels). It was also clearly established that, in light of the initial data available (i.e., chest pain presentation, history, and initial ECG), this determination should be based on

risk. Basically, these five levels represent the relative risks of acute MI or ongoing acute ischemia in association with acute coronary syndrome. Levels 1 and 2, the higher risk levels, did not involve the introduction of any new technologies but simply systematized the available data from presentation and initial ECG into critical pathways for the treatment and disposition of these patients. Level 1 is the highest risk population and constitutes those patients who meet thrombolytic criteria by ECG on presentation. The critical pathway provided for early revascularization either by thrombolytics or primary percutaneous transluminal coronary angioplasty. Level 2 patients were also designated according to their initial ECG. For those patients presenting with ischemic ECG changes, the critical pathway provided for direct admission to the coronary care unit, with subsequent use of serial markers, ECGs, and monitoring to document the presence or absence of the acute coronary syndrome. In most cases, invasive or noninvasive diagnostic testing followed. At the other extreme, level 5, constitutes the patients whose presenting chest pain is clearly noncardiac in nature and who can be given a definitive diagnosis.

Clearly, the three levels mentioned so far do not encompass the patients who are most problematic to the ED physician making triage decisions. The moderate risk category, levels 3 and 4 in our system, often represents difficult triage decisions. In our scheme, level 3 is for the patient with probable unstable angina. Generally, this is the patient who presents with typical symptoms lasting greater than 30 minutes and a normal ECG or with atypical symptoms and a nondiagnostic ECG. The level 3 patient is admitted to a fast-track "rule-in" unit where serial markers and ECGs are used to rule-in acute MI or acute coronary syndrome. In addition, the level 3 patient is injected with a tracer dose of technetium-99m sestamibi (MIBI) in the ED as soon as triage assignment is made. Subsequently, the patient undergoes imaging, and the results of the single photon emission computed tomography (SPECT) perfusion imaging are used to help determine the need for early intervention—or in the case of the normal study, for early noninvasive stress testing before discharge. Most patients, however, fall into an even lower risk category, with typical symptoms lasting less than 30 minutes and a normal ECG or with atypical symptoms of short duration and a nondiagnostic ECG. Although the risk of acute MI is extremely low in this group (estimated at less than 5%), the risk of acute coronary syndrome is higher, creating a significant risk for the early discharge of these patients. In the ACT strategy, patients falling into this category (level 4) receive a tracer dose of MIBI in the

ED and undergo myocardial perfusion imaging for the primary purpose of risk stratification rather than diagnosis. As a risk stratification tool, the imaging provides evidence for unsuspected acute coronary syndrome or acute MI and may lead to early revascularization of the patient who is otherwise believed to be at low risk. The patient who has an entirely normal study may be admitted to a noncoronary intensive care unit area or (in most cases at our institution) sent home and scheduled to return for stress testing within the next 48 to 72 hours.

The basis for disposition of the low-risk (level 4) patient is found in the Agency for Health Care Policy and Research (AHCPR) guidelines on unstable angina.¹⁹ The two components of the AHCPR evaluation strategy are the determination of the presence or probability of underlying coronary artery disease (CAD) and the assessment of risk for hard cardiac events. When both are determined to be low, the guidelines suggest that it may be appropriate to use outpatient follow-up evaluation within 72 hours of the index encounter. By definition within the ACT strategy, patients placed in level 4 are believed to be at low risk for significant underlying CAD. However, the probability of a subsequent adverse outcome cannot be predicted from the initial evaluation with the same degree of certainty. It is well established that the presence or absence of risk factors, although predictive of underlying CAD, may not be predictive of an acute event.²⁰ We also know that the presenting ECG may not detect up to 40% of acute MIs, especially in the posterior distribution.^{5,21} These observations appear to confirm that, although CAD and acute syndromes are definitely interrelated, they do not necessarily overlap. Significant CAD is a chronic condition upon which the dynamic process of acute coronary syndrome may be superimposed. However, the acute coronary syndrome may occur in what was considered anatomically insignificant coronary disease. This leads us to ask what techniques are available to predict the presence of the acute coronary syndrome. In reviewing the pathophysiology of acute coronary syndrome, we know that the initial event is commonly plaque rupture, which leads to intracoronary thrombosis, reduced blood flow, ischemia, and eventual myocardial necrosis or infarction. The optimum strategy would allow us to predict impending plaque rupture or intracoronary thrombosis, but no clinical tools that would be useful in the ED setting are currently available. However, noninvasive tools are available for evaluation of the later portions of the cascade of events, reduced blood flow and myocardial ischemia. We know that myocardial perfusion imaging allows us to detect acute blood flow reductions even in the

resting state²² and that echocardiography can demonstrate wall motion abnormalities resulting from ischemia.^{23,24} In addition, newer myocardial perfusion imaging agents, such as MIBI, enable simultaneous evaluation of both perfusion and function, giving a more global assessment of the status of the ventricle.^{25,26} The premise, as supported by early studies, is that a normal perfusion/function study indicates a low risk for subsequent adverse cardiac events, at least in the short term (i.e., the first 72 hours).^{27,28} Therefore, the second component of the unstable angina assessment appeared to be met by the use of acute perfusion imaging in the ED.

We evaluated the power of acute perfusion imaging as a predictor of outcome in a one-year follow-up study involving 1,187 patients, of whom 438 were assigned to levels 3 and 4 and underwent acute perfusion imaging.²⁹ A comparison of MI and revascularization between patients with normal and abnormal perfusion imaging revealed highly significant differences. During their initial admissions, the patients with normal studies experienced no acute MIs and seven revascularizations, whereas the patients with abnormal studies experienced seven MIs and 30 revascularizations. On 1- to 12-month follow-up in the same population, the results of the acute perfusion imaging study continued to be predictive of outcome. Over the subsequent 12 months, four additional MIs, two additional revascularizations, and eight cardiac deaths occurred in the group with abnormal studies. No late MIs, three late revascularizations, and no cardiac deaths occurred in the group with normal studies. In a subsequent comparison of MI, revascularization, and imaging results among the patients initially triaged to levels 2, 3, and 4, we observed no significant difference in outcomes between patients assigned to level 2 (a high-risk group with presenting ischemic changes on ECG) and those assigned to the lower risk groups who had an abnormal SPECT MIBI study during their index ED evaluation. In other words, the patient who is initially triaged to low risk with a normal or nondiagnostic ECG but has an abnormal acute perfusion study in the ED has the same outcome risk as the patient presenting with ischemic changes on ECG. There are now three studies in the literature, comprising a total of 604 patients who were evaluated with acute perfusion imaging in the ED setting.²⁷⁻²⁹ Although the total patient population is still relatively small, all of the 32 patients determined to have MI were identified by the perfusion imaging study, for 100% sensitivity; on the other hand, none of the 442 patients with normal studies had an acute MI, for a negative predictive value of 100%. In reviewing total cardiac events (hard and soft), the negative predictive value was 97.5%.

As suggested by these results, acute myocardial perfusion imaging in the ED may help us achieve two of the four goals: reduction in the frequency of soft admissions and in the probability of inappropriate disposition. Yet it may also help us meet the first goal: reduction in delay to therapy and disposition. Clearly, among low-risk patients with nondiagnostic ECGs, a significant high-grade perfusion defect indicates the presence of a high risk zone and the need for intervention. Thrombolytic data indicate that time is an important element in myocardial salvageability;³⁰⁻³² therefore, it appears that early identification of significant risk zones provides the opportunity for early intervention and optimal benefit in this group of patients. Among the level 4 patients in the ACT study, seven underwent early revascularization, but only two "ruled-in" for acute MI. Although this benefit is unlikely to be reflected in a conventional cost-effectiveness evaluation, it may be one of the greatest values of the technique, because early intervention in the patient with a high risk zone may significantly reduce morbidity and mortality, leading to reduced costs in both the short and long run.

Our fourth goal is to reduce overall cost and increase efficiency. In the current health care environment, a resultant improvement in the quality of care may not be sufficient to justify new strategies or technologies. Technologies related to cardiovascular disease involve even greater concern over cost due to the widespread prevalence of disease and the global cost. In 1992 the National Hospital Ambulatory Medical Case Survey by the National Heart, Lung, and Blood Institute estimated that 5.5 million chest pain patients present to EDs yearly, representing 6% of all ED visits and over 10% of ED admissions. In 1987, the AHCPR estimated that U.S. hospital costs for ischemic heart disease exceeded \$13 billion. More recently, in 1993, the National Heart Attack Alert Program of the National Institutes of Health estimated that U.S. hospital costs for low risk patients exceeded \$4 billion.

Two published studies have addressed the cost-effectiveness of using acute perfusion imaging in the evaluation of chest pain patients. The first study used a survey technique in 50 patients with no prior MI, nondiagnostic ECG, and a low risk history and physical.³³ The patient was evaluated in the routine fashion and a disposition was planned. Then the patient underwent imaging and, with the imaging results included in the decision process, another disposition was planned. Using this technique, the authors estimated a cost reduction of approximately \$786 per patient. Interestingly, disposition changed to home in 54% of patients. In a recently published larger and more sophisticated study, researchers compared "NO SCAN" versus

"SCAN" strategy in a total of 209 patients: 102 underwent the SCAN strategy and 107, the NO SCAN strategy.³⁴ All patients presented to the ED with chest pain and had a normal or non-diagnostic ECG. In the NO SCAN category, patients who were considered high risk on the initial evaluation were admitted, whereas the low-risk patients were discharged. In the scan strategy, patients with an abnormal or equivocal imaging study were admitted, and those with a normal study were discharged home. The authors used both Medicare and institutional source data to calculate mean, median, and simulation costs. In all analyses, the SCAN strategy was significantly less costly. Using mean costs from Medicare data, the SCAN strategy was 17% less (median 10%), and using institutional resource data, it was 14% less (median 7%). Interestingly, the mean savings were \$1,032 using Medicare data and \$796 using institutional data; these numbers are quite similar to the savings reported in the survey technique previously mentioned. Probably the most important conclusion made in the latter cost study resulted from a sensitivity analysis, which revealed that the NO SCAN strategy would be less costly than the SCAN strategy only when the incidence of adverse events in patients presenting to the ED with chest pain and a nondiagnostic ECG exceeded 60%.

We have recently completed an institutional resource-based cost analysis in a total of 1,488 patients, 879 of whom were enrolled in the ACT protocol group and 609 in a control group. The results from this study have not yet been published, but they demonstrate cost savings similar to the previously reported studies. This study also documented decreased lengths of stay, both total and in critical care units, and a reduction in the admission rate of approximately 15% compared to the control group. It is encouraging that each of these three cost-effectiveness studies used a different methodology, yet all three have demonstrated similar cost savings.

In conclusion, we feel that the ACT strategy is a powerful example of the potential of multispecialty, process-oriented health care. Using the expertise of a multispecialty team to address the entirety of the patient care process and using specific diagnostic testing in the decision analysis allowed us to achieve substantial gains toward all four goals. We believe that multispecialty process-oriented health care may be one of the most significant positive opportunities that has inadvertently resulted from the health care reform movement.

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The early diagnosis and treatment of non-ST-segment elevation acute ischemic syndromes: the need for a new approach

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ABSTRACT: *The diagnosis and treatment of ST-segment elevation myocardial infarction (MI) has been well defined by many multicenter trials. However, the treatment and diagnosis of non-ST-segment elevation MI is much less understood. Single photon emission computerized tomographic perfusion imaging shows great promise in risk-stratifying patients into low-risk and high-risk subsets when 12-lead ECG and initial cardiac enzymes are normal or nondiagnostic. Early exercise stress testing may be important in further risk-stratifying patients whose chest pain has resolved or who have low risk perfusion scans in the emergency department. The role of two-dimensional echocardiography in non-ST-segment elevation MI is currently under investigation.*

Limitations of the ECG

The electrocardiogram (ECG) has been the mainstay in the screening of patients for acute coronary syndromes. The clinical diagnostic problem, however, is that the ECG is only diagnostic in a minority. The earliest signs of acute myocardial infarction (AMI) elevation of the ST segment >1 mm is seen in only 40% to 60% of patients presenting with an AMI.¹ In the GUSTO AMI Registry, a large portion (36%) of patients presenting with myocardial infarction (MI) had no ST elevation and had over three times the mortality (17%) as patients with ST elevation who were treated with thrombolysis (5%).² The challenge is to develop better diagnostic tools to identify patients who present with MI without ST-segment elevation so that therapy may be initiated more promptly.

Clinical effectiveness. Injection of 99mTc-sestamibi in patients with nondiagnostic ECGs and chest pain has demonstrated high sensitivity in detection of coronary artery disease (96%)³ and subsequent cardiac events (94%).⁴ Gregoire and Theroux demonstrated that acute myocardial perfusion imaging with 99mTc-labeled sestamibi, when injected during chest discomfort, was significantly more sensitive than 12-lead electrocardiography. The 12-lead ECG was only 35% sensitive in detecting coronary artery disease during chest pain, whereas 99mTc-sestamibi SPECT was 96% sensitive.³ Therefore, myocardial perfusion imaging is a better tool in detecting myocardial ischemia than the 12-lead ECG. In a study by Hilton and Stowers,⁴ 99mTc-sestamibi SPECT was superior to the ECG alone or in combination with clinical variables in the prediction of subsequent cardiac events. A negative scan was associated with <2% incidence of in-hospital cardiac events, and a positive scan with >70% incidence of in-hospital cardiac events.

Cost effectiveness. Another reason for early risk stratification with myocardial perfusion imaging is the economic issue of the high rate of hospitalization of low-risk patients. At St. Luke's Hospital, before the use of sestamibi was implemented, patients with suspicious chest pain and a nondiagnostic electrocardiogram had a 96% admission rate. However, after the implementation of myocardial perfusion imaging with 99mTc-sestamibi, the admission rate fell to around 60%. In our study, the clinical decision to admit or discharge the patients was based on an abnormal but nondiagnostic electrocardiogram or more than three risk factors versus a positive sestamibi scan. In comparing these two strategies, we demonstrated a 17% reduction in hospital cost, with a \$1,032 per patient savings in the group undergoing sestamibi scanning versus the group undergoing clinical risk assessment with electrocardiography.⁵

In the 3- to 12-month follow-up of 180 patients, there were no subsequent cardiac events at three months in the patients who were discharged from the emergency department with a negative scan.⁶ Therefore, 99mTc-sestamibi appears to be safe and highly practical in the emergency department setting. It separates patients at intermediate risk from very low- and high-risk groups. It allows for expeditious cardiac evaluation, and a negative scan is associated with excellent short-term prognosis at three months.

The release of plasma MB CK occurs within 40 to 60 minutes of sustained occlusion, but the extent and rate of release is minimal such that plasma CK levels remain normal for up to 6 to 8 hours.⁷ Cardiac troponins have recently shown promise in predicting subsequent cardiac events in patients who present with chest pain, but again it takes several hours after coronary occlusion for plasma levels to become abnormal.⁸ This delay in elevation of cardiac enzymes limits their value in directing coronary intervention in patients with non-ST-segment elevation MI.

Two-dimensional echocardiography

Two-dimensional echocardiography has also been shown to be effective in risk stratification of patients with chest pain and a nondiagnostic electrocardiogram, but has a lower sensitivity (88%) and specificity (78%) than 99mTc-sestamibi imaging.⁹ However, patients with normal left ventricular function and absence of wall motion abnormalities on two-dimensional echocardiography have an excellent short-term prognosis.

Early exercise stress testing

Early exercise stress testing can also be used to risk-stratify patients with chest pain and a nondiagnostic electrocardiogram in the emergency department. Results of the ROMIO study identified lower cost for patients in the emergency department who undergo early exercise stress testing; however, the group in whom myocardial infarction was ruled out rapidly still had a mean hospital stay of 15.1 hours.¹⁰

Although early exercise stress testing in this group has been proven to be safe and cost-effective, not all patients can perform adequate exercise. In the Duke Treadmill Study, 54% of patients were still in the intermediate-risk group after completion of the treadmill exercise stress testing.¹¹ This group of patients at intermediate risk will require further risk stratification.

Injection of 99mTc-sestamibi in patients whose chest pain has resolved is associated with a significant lowering of sensitivity in the detection of coronary artery disease (96% sensitivity in patients with symptoms compared with 64% sensitivity in patients with resolved chest pain).³ Therefore, it is important that negative scan patients injected following symptom resolution be risk-stratified further with exercise stress testing. At St. Luke's Hospital, a standby dose of 99mTc-sestamibi is available for injection during exercise stress testing if it is felt the patient will still be at intermediate risk after completion of exercise.

During the past decade, the treatment of acute ST-segment MI has dramatically improved, with the early initiation of aspirin, thrombolytic therapy, and primary percutaneous transluminal coronary angioplasty. The next major step in improving care of acute coronary disease will be in the early diagnosis and treatment of patients who present without ST-segment elevation MI. The SMAC study is designed to look at the value of cardiac troponins, Tc-99m tetrofosmin SPECT perfusion imaging, and two-dimensional echocardiography in the diagnosis and treatment of non-ST elevation MI. In the 50-patient pilot trial, patients with chest pain and non-ST-segment elevation will be risk-stratified on the basis of the SPECT perfusion image to early catheterization and revascularization (high-risk scan) and early exercise stress testing and discharge (low-risk scan). It is hoped that close collaboration by cardiologists and emergency department personnel in this study will lead to a better understanding of the best approach to diagnosis and treatment of patients who present with chest pain without ST elevation.

Conclusion

A more rapid method of triaging emergency department patients with chest pain and non-ST-segment elevation is long overdue. The cost of care for patients who are admitted with chest pain but do not have myocardial infarction is estimated at 13 billion dollars annually.¹² What has been an acceptable approach in the past will not be an acceptable approach in the future. We must be ready to take advantage of new diagnostic techniques, and certainly myocardial perfusion imaging appears to be one of the most promising. However, to be cost effective and clinically effective, SPECT perfusion imaging should be applied early to the appropriate intermediate-risk patients. This will allow early intervention in high-risk patients and early discharge in low-risk patients. Only a philosophy similar to that of cardiac catheterization laboratories with standby doses ready to be immediately injected, technicians on call to rapidly image, and experienced physicians available for accurate and timely reading of SPECT perfusion imaging will ensure its future success. It is possible that in the future, nuclear cardiac imaging will prove to be most valuable when applied early to risk-stratify patients with acute ischemic syndromes in emergency departments and chest pain centers.

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Implementation of an acute myocardial perfusion imaging program for patients with chest pain and nondiagnostic electrocardiogram: the St. Agnes experience

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ABSTRACT: *One of the current focuses of the chest pain emergency department (CPED) movement is to improve community awareness of chest pain signs and symptoms and thereby encourage patients to be evaluated early. However, such a strategy may overwhelm the mechanisms currently in place to evaluate these patients in a timely and cost-effective manner. In this article, the author reviews the experience of St. Agnes HealthCare using acute myocardial perfusion imaging (MPI) to help stratify patients who present with chest pain and nondiagnostic or normal ECG into low- and high-risk groups for the development of acute cardiac events. Currently, St. Agnes is participating in a randomized, multicenter trial to assess clinical and cost efficacy of employing a strategy of acute MPI in the CPED.*

Emergency department (ED) evaluation of patients presenting with chest pain has traditionally involved history, physical examination, electrocardiogram (ECG), and cardiac enzyme evaluation.¹ Unfortunately, these methods suffer from suboptimal sensitivity and specificity for adequate timely triage of many patients, particularly those with atypical symptoms and/or nondiagnostic ECGs.

The current strategy is to admit patients with chest pain of suspected cardiac origin to a monitored bed for precautionary reasons. Typically, the hospital stay lasts 24 to 48 hours and is followed by a provocative stress test before discharge. This strategy poorly utilizes limited medical resources because most of these patients do not suffer acute myocardial infarction (AMI). Despite the low threshold for admitting patients with chest pain, as many as 5% to 8% of patients discharged from the ED are found to have suffered an AMI.²⁻⁴ In fact, failure to diagnose and treat AMI patients in the ED accounts for the largest settlements of malpractice lawsuits.⁵

Since the advent of thrombolytic therapy and acute angioplasty for intervention in patients suffering AMI, there has been a dramatic decline in mortality for patients able to reach the ED (5%). Unfortunately, the mortality rate for patients who suffer AMI without meeting the criteria for acute intervention (i.e., those without ST-segment elevation or bundle branch block) has not changed significantly and remains three times higher (17%).⁶

Therefore, there is a need to find a new diagnostic strategy to stratify chest pain patients who present to the ED according to level of risk for the development of cardiac events. For example, low-risk stratification would facilitate early ED discharge and conserve precious resources, while the accurate identification of a high-risk patient subgroup would allow early initiation of appropriate therapy and hopefully reduce mortality.

Methods and materials – The St. Agnes experience

St. Agnes HealthCare, a 400-bed community hospital located in Baltimore City, Maryland, is home to the nation's first chest pain emergency department (CPED) with approximately 4,000 visits per year. In 1993, we began a pilot study to determine whether acute myocardial perfusion imaging (MPI) would complement the services of our busy CPED. Our initial results from 28 patients showed a sensitivity and specificity for predicting cardiac events (i.e., MI, cardiac death, or need for revascularization) of 100% and 84%, respectively.⁷ These results, together with other published reports, led us to conclude that acute MPI was a useful tool for evaluating patients with chest pain and nondiagnostic ECG.

Our program initially studied patients during the working hours of the nuclear medicine department. As the ED and cardiology department became more comfortable with the reliability of the data acquired from our institution there was a greater need to expand this service. An in-hospital technologist position was expanded to 9:30 p.m. for injection and scanning of patients. Later in the evening, the ED physician would come to the nuclear medicine department to get a dose of radiopharmaceutical for injection and a technologist would be called in to scan the patient. Patient data was promptly transmitted via computer modem to the physician's home for interpretation.

Arrangements were made with our local radiopharmacy provider, Syncor International Corporation, to provide a standing order of the radiopharmaceutical to have available around the clock. By paying a small fee, doses may be returned to the radiopharmacy for full credit, thus making the program fiscally sound.

In late 1996, the nuclear medicine department purchased two variable angle gamma cameras and comput-

ers. We now routinely perform gated tomographic acquisitions on all patient studies. This capability allows the evaluation of not only perfusion, but also provides accurate assessment of left ventricular thickening, regional and global wall motion, as well as ejection fraction.⁸

Beginning June 1997, St. Agnes is participating with five other centers in a prospective randomized clinical trial to assess clinical and cost efficacy of sestamibi imaging in the ED. This study will be the largest of its kind with a projected enrollment of 5,000 patients. Patients will be randomized to a strategy of either "no scan" (current diagnostic workup) versus "scan," where results of sestamibi imaging will be included in the diagnostic workup. Results of such studies are necessary to provide data to recommend employing acute MPI as the standard of care in the workup of the chest pain patient with nondiagnostic ECG. The current lack of prospective randomized trials in the literature has led the National Heart Attack Alert Program to decline to endorse routine acute MPI at this time.⁹

A total of 627 acute MPI have been performed on ED patients presenting with chest pain and nondiagnostic ECG at our institution through the end of 1996. The sensitivity, specificity, and accuracy rates for the prediction of cardiac events is 96%, 82%, and 84%, respectively.

Discussion – radionuclide perfusion imaging

Multiple studies have confirmed the utility of early imaging of ED patients who present with chest pain and nondiagnostic ECG. Bilodeau et al.¹⁰ demonstrated that injecting symptomatic patients with 99mTc-sestamibi was highly sensitive for the detection of coronary artery disease (96%), while the ECGs of these patients were less sensitive (35%). Varetto et al.¹¹ showed that MPI had a high negative predictive value for the exclusion of cardiac events in a cohort of patients presenting to the ED with chest pain and nondiagnostic ECG over an 18-month follow-up period. Hilton and Stowers¹² demonstrated the ability of MPI to stratify ED patients into high- (>70%) and low- (<2%) risk groups for in-hospital cardiac events which was superior to ECG and clinical variables. Tatum et al.¹³ noted that the sensitivity of MPI for detection of AMI was 100%. During a one-year follow-up period, patients with a negative scan had an event rate of 3% (revascularization), but suffered no AMI or death.

Two studies have addressed cost-effectiveness of employing a strategy which includes MPI. Weissman et al.¹⁴ retrospectively reviewed 50 patients who underwent MPI from the ED. They concluded that the total savings of using MPI was \$786 per patient which came mainly from the ability to directly discharge 29 of the patients home.

Radensky et al.¹⁵ compared a cohort of ED patients who underwent MPI, versus a group who did not. Whether using Medicare or institutional data, the group using MPI was more cost-effective (\$1,032 versus \$796 savings per patient).

Summary

The process of developing an acute myocardial perfusion imaging program has been a challenge for multiple departments of the hospital including CPED, cardiology, nuclear medicine, nursing, coronary care unit, transportation, and administration. However, each has worked together and provided the necessary input and feedback to create a successful program. Each has been an integral component of the process. This multidisciplinary program was well received by the last hospital review by the Joint Commission on Accreditation of Healthcare Organizations.

Although the initial focus of the process was to reduce unnecessary admissions through the CPED to monitored beds, it appears that early identification of patients without ECG changes who have ischemic or infarcting myocardium may help reduce the mortality rate in this group. Since St. Agnes does not perform coronary angioplasty, a number of our patients with positive scans have been successfully transferred directly to other institutions from the CPED for acute interventions.

One of the current focuses of the CPED movement is to improve community awareness of chest pain signs and symptoms and thereby encourage patients to be evaluated early.¹⁶ Many of these patients will present to emergency departments throughout Maryland, but will the mechanisms be in place to adequately evaluate them without taxing the current system? It is this author's opinion that acute MPI can provide the mechanism to stratify high and low risk patients in a timely and cost-effective manner.

What then becomes the best clinical practice? Maryland physicians must realize that the old paradigm of admitting all patients with chest pain and nondiagnostic ECG is neither good patient care nor cost-effective. The challenge is to develop acute MPI programs which have competent technologists and physicians available for rapid patient throughput and scan interpretation. If ongoing prospective clinical trials confirm the experience of chest pain centers currently performing MPI, it is likely that MPI will become the standard of care for the evaluation of ED patients with chest pain syndromes.

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Full text of “Current status of sestamibi in the risk stratification of chest pain patients: a panel discussion” with Drs. Spiegler, Stowers, and Tatum, is available on the web site at www.chestpaincenters.org.

Early Heart Attack Care on the World Wide Web

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ABSTRACT: *The Internet is an excellent medium for storing and quickly disseminating large amounts of information on a global basis. The ability to query information “storehouses” makes it even more powerful for end users. This brief article demonstrates key features of the Early Heart Attack Care (EHAC) Web site. Basic navigation and search techniques are covered. The EHAC site contains one of the largest, if not the largest, collection of early heart attack care materials. It can be accessed easily, from virtually anywhere in the world, via the Internet using most standard Web browsers. The World Wide Web, with its graphic multimedia interface, is ideally suited for EHAC’s mission of worldwide dissemination of early heart attack care concepts and information to both professionals and the community.*

Disclaimer: *The Infobahn is still work in progress. It is not uncommon to hit traffic jams and dead-ends. We have tried to verify most links to the EHAC site but cannot assure their future maintenance.*

The original EHAC site address, www.ehac.org, provided access to a single site. As a result of feedback from users, it is now split into two sites: a clinical site with the address www.chestpaincenters.org and a consumer site with the address www.chestpain.org.

How to use the EHAC sites

The choice of which EHAC site one visits depends upon whether one is a provider or a consumer. A provider would most likely visit the clinical site first, while a lay person is more likely to find relevant information at the consumer site. Each site provides an index of the site’s entire contents.

The main screen at the clinical site categorizes information in logical

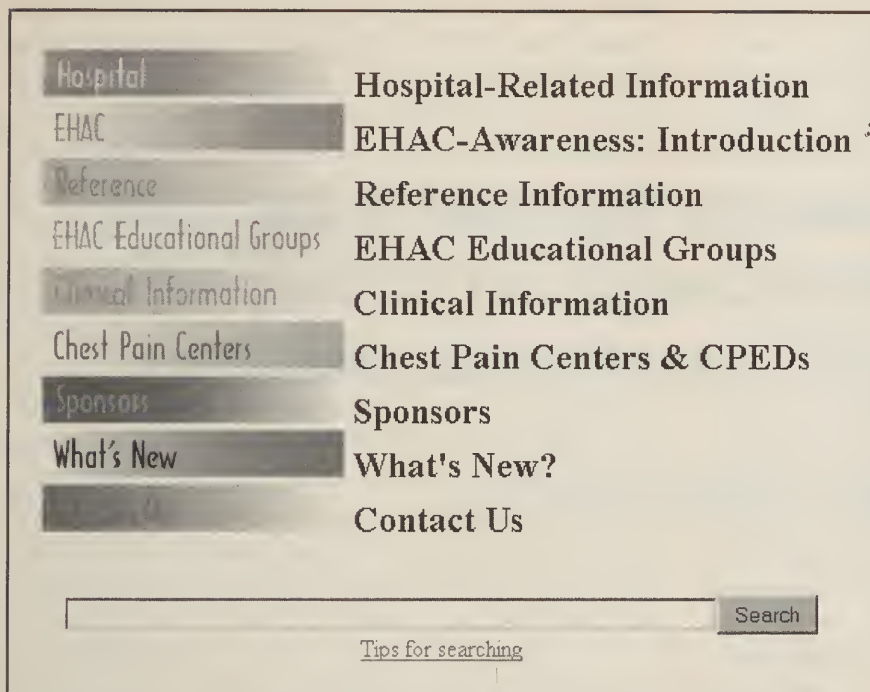


Figure 1.

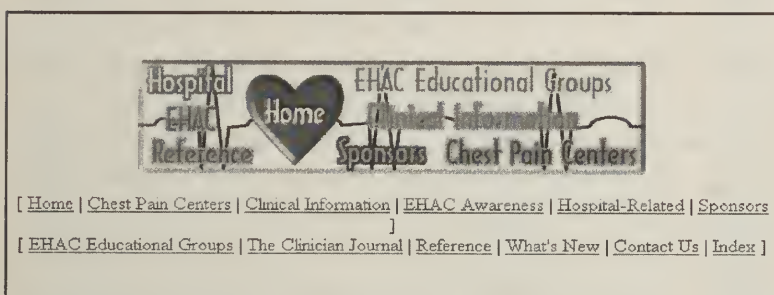


Figure 2.

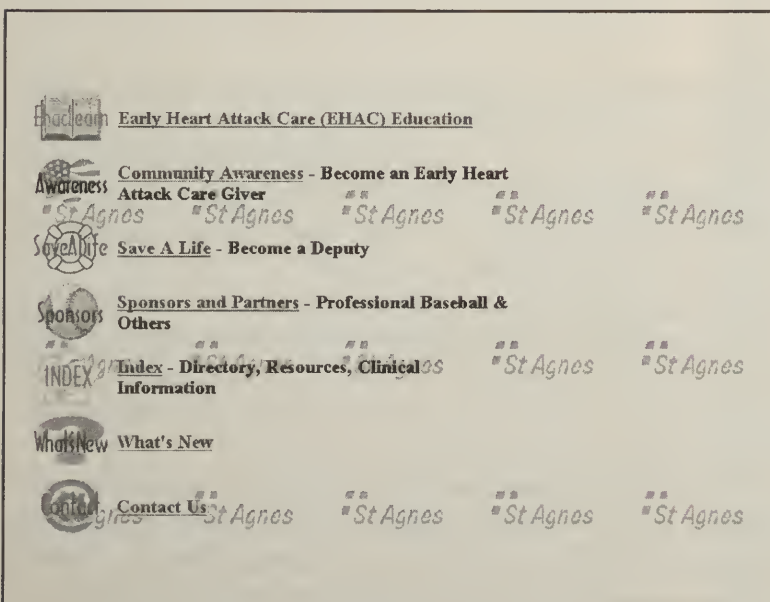


Figure 3.

groups (Figure 1). This menu is most useful for searching information by category (e.g., if the information sought is hospital related, going into the hospital section may provide the quickest access). It is, however, impossible to categorize all information in intuitive categories.

The search box at the bottom is useful when a user is unable to determine where to start. It allows a search of the entire site by entering key words or a string of plain English text. Choice of words is important for quality search results. The more specific the words used to describe an area of interest, the more likely a correct match will result. Results are displayed as a list of the closest matches with hyperlinks to the sources. Double clicking on the hyperlinks opens relevant documents,

pages, or other sites. Another way of searching is through the site index, which can be accessed from the index link at the bottom of each page. This provides a simple alphabetical link to all contents of the site. Extensive external links are provided in the reference section. The bottom of each page also contains links back to the home page and other categories (Figure 2).

The consumer site organizes information for nonmedical or lay people (Figure 3). A wealth of educational information, from becoming an early caregiver to becoming a deputy, is provided in this section.

How to communicate with EHAC

Both the clinical and consumer home pages have clearly marked links to e-mail forms, which are a quick and simple way of communicating with EHAC staff. ■

This brief introduction is simply to get you started. It obviously can not substitute for the real online experience. Happy surfing.

Can Florida become the first state in the United States to take heart disease out of first place?

The Maryland versus Florida challenge

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ABSTRACT: *The high morbidity and mortality from acute myocardial infarctions (AMI) prompted Dr. Raymond Bahr to challenge the author as to whether Maryland or Florida could be the safest state in which to have an AMI. He extended this competition spirit to a second debate as to which state would first take heart disease out of first place as the cause of death. Such a goal appeared laudable. But is it? Would AIDS, tuberculosis, or widespread cancer be a better way to die? Thinking not, Florida cardiologists established a goal to become the safest state, with the nation's largest percent of physiologically active centenarians, who would eventually die peacefully, even if due to a heart attack.*

At first glance, taking heart disease out of first place as the cause of death for Florida's citizens would appear to be a highly desirable goal. During the last three or more decades, it is a goal that many thoughtful physicians and other health care providers have been working diligently to attain. It is a goal that should be attainable, for heart disease has not always been the most common cause of death in our society.

In 1910, coronary artery disease (CAD) caused only 10% of the deaths in the United States; tuberculosis and other infectious diseases were by far the most common cause.^{1,2} But by 1948, CAD accounted for 50% of the deaths in our society, and tuberculosis and other infections were becoming less common causes.

The incidence of death from CAD throughout America continued to increase until 1962 when it peaked at 56.1%.¹ Since that year, the incidence of death from CAD has been gradually declining. Like the earlier increase in the incidence of death, most attribute this decline to increasing numbers of our society abandoning adverse lifestyles that

encourage the development of CAD and more people actively pursuing a heart-healthy lifestyle that encourages a decreased incidence of CAD. In 1984, Goldman and Cook³ reported that 62.5% of the reduction in premature death from CAD was due to a reduction in the hazards of elevated cholesterol, smoking, and hypertension; most investigators attribute this decline to controlling these same health hazards; some investigators also emphasize the importance of correcting obesity and a sedentary lifestyle.^{4,5}

This emphasis on change of lifestyle is not intended to discount the importance of carefully timed and appropriately selected medications, instruments, and/or techniques to be used when CAD threatens to cause acute or life-threatening changes. To benefit from such efforts, the leaders of the Florida Chapter of the American College of Cardiology (FCACC) developed a program to make Florida the safest state in which to have an acute myocardial infarction (AMI).

The FCACC established this goal in August 1994 at the Consensus Development Conference held at St. Joseph's Hospital in Tampa, Florida. This conference was attended by representatives of 13 national and state organizations whose members are actively involved in AMI patient care in Florida.^{6,7}

In 1992, 96.7/100,000 Floridians died of an AMI. This ranked Florida 25th from the lowest among the 50 states and the District of Columbia in the incidence of deaths from AMI; New York had the highest incidence with 126.9/100,000 and Hawaii the lowest with 58.0/100,000. Regardless of significant differences in the age of the populations in these states, calculating the mortality as age-adjusted equalized the incidence of death per state. Florida's goal is to exhibit the lowest incidence of death from an AMI by the year 2000 (or shortly thereafter). In view of the friendly competition between Florida and Maryland, it is important to note that Maryland had the 7th lowest age-adjusted mortality from an AMI among the 50 states in 1991.

In order to make Florida the safest state in which to have a heart attack, 20 recommendations for action were adopted by consensus at the St. Joseph's Conference. These recommendations, including appropriate discussions and a review of the pertinent literature, were published as a single issue of the *Journal of the Florida Medical Association* in February 1995.⁶⁻¹¹

Much progress has been made to attain this goal. From the start, an effort was made to insure that emergency trained physicians staffed the emergency departments (EDs) of hospitals throughout Florida.

A Florida citizen experiencing an AMI in a metropolitan area would normally receive the most advanced treatment for

CAD in a nearby metropolitan medical center. But should that person experience a heart attack while visiting a remote rural community, the care might be less than ideal. The life (and quality of life) for that person would be dependent on the care he or she receives in this remote area. For this reason, we gave priority to the development and maintenance of a seamless and enhanced 911 system, using two-way communication and a mandated statewide emergency medical dispatch system.¹¹

In 1992, one-third of U.S. residents did not have access to 911, and 12 counties in Florida did not have such a system.⁹ More than 1,000 Americans experienced a cardiac arrest each day and 95% died.¹² Furthermore, nearly one-half of the 500,000 CAD deaths occurred suddenly. Many lives could be salvaged by the immediate availability of an automated external defibrillator (AED) and readily available personnel knowledgeable in the use of such a device. But many other factors determine the success of resuscitation of a sudden death victim.

In 1991, only 1.4% of sudden death victims in New York City survived, whereas 29% survived in King County (Seattle), Washington.¹³ In part, this difference depended on six factors: rapid recognition of the seriousness of the situation, how quickly help was sought, the knowledge and skill in maintaining an airway and circulation during early attendance, the rapid response of the EMS vehicle, the knowledge and skill of the EMS personnel, and the equipment available for defibrillation.

Florida is fortunate that the University of Miami School of Medicine has established the Medical Training and Simulation Laboratory. This is a unique training facility directed by Michael Gordon, M.D. Dr. Gordon designed a training program for paramedic personnel that teaches them the urgent management of trauma and heart attacks. Nearly 3,000 paramedics per year graduate from his program. Plans are under way to obtain support from the Florida State Board of Regents, the Department of Health, and municipal and county emergency medical systems (EMSs) to make this program available through junior colleges statewide.

As soon as the focus shifted to early treatment of AMI, we became concerned about the shortage of well-trained, dedicated-to-duty, emergency medicine physicians in Florida and across the nation. At the same time the FCACC was conducting the Consensus Development Conference in Tampa, the Josiah Macy, Jr., Foundation sponsored a conference to define the role of emergency medicine in the future of American medical care.¹⁴ This was a very probing conference.¹⁵ It was concluded that although EDs are widely available, they vary considerably in quality and accessibility from region to region (and in many regions from neighborhood to neighbor-

hood). Such variability in the quality of care poses a major dilemma for the physician who urges patients to seek out an ED when they experience chest pain.

At that conference, L. Thompson Bowles, M.D., president of the National Board of Medical Examiners, and conference chairperson, reported that, “for too many emergency rooms in America, a stricken patient cannot be sure he or she will be treated by a qualified, competent emergency physician.”

In addition, many medical schools in this country do not devote attention to training the physicians of tomorrow to respond to emergency situations. Only seven medical schools have a required emergency medicine rotation for an average of only three weeks in the third year of training. An additional 14 schools have such a required rotation during the fourth year. Although 81% of the students are required to be skilled in basic life support to graduate, only 43% have a requirement for competency in advanced life support; only 52% of graduating students must know how to start an intravenous line, and only 39% must know how to intubate a patient.

At the Macy Conference, it was stated that “when people ask if there is a doctor in the house, they have reason to expect that every physician can do the minimum to save a person’s life in an emergency. This is not the case today.” However, students who graduate and obtain a license to practice are actively recruited to “moonlight” in many EDs in this country.

This shortage of adequately trained emergency physicians is a cause for concern in Florida. In 1995, there were only two approved emergency medicine training programs in the entire state, graduating only 21 physicians qualified to apply for American Board of Emergency Medicine certification. Additional programs are under development. The FCACC is urging state support for them, for the shortage is critical.

As previously indicated, the incidence of death from CAD in the United States has been declining steadily for the last three or more decades. This is so because increasing numbers, albeit only a small number of individuals throughout the entire population, are pursuing a more heart-healthy lifestyle. They have accepted what has long been recognized: “The road to wellness, although a life-long journey, holds benefits along the way for those who seek prevention before they seek cure.”¹⁶

If Florida is to beat Maryland in the current challenge, this message must be expressed to many more Floridians; the incidence of death from CAD in Maryland is already far lower than in Florida. So, who will teach our society desirable preventive, heart-healthy practices? Who will see that such practices are widely adopted? Clearly it should be the doctor.

The responsibility of the doctor to be a teacher dates back to the days before Hippocrates. The word “doctor” comes from the Latin word *docere*, which means “to teach.” Unfor-

tunately, very few doctors devote meaningful time trying to teach their patients how to abandon adverse health habits and adopt heart-healthy ones.

Robert Levy, M.D., when director of the National Heart, Lung and Blood Institute (NHLBI), said: “We learned from the public that they feel less than half of the physicians spend any considerable time providing them with information dealing with prevention.”¹⁷ J.C. LaRosa, M.D., reported that cholesterol screening is done in no more than about one-third of patients who visit doctors.¹⁸ Furthermore, according to Eugene Braunwald, M.D., “there are about seven million survivors of acute myocardial infarction in the United States and the vast majority are not receiving lipid-lowering therapy.”^{19, 20}

The cholesterol levels of AMI patients decline rather markedly shortly after an acute myocardial event, and the level does not become stable for about three months. As a result, many primary care physicians and cardiologists postpone attention to the lipids “until later.”^{20, 21} But “if and when later arrives,” the patient is “back in a routine,” hopefully without symptoms, and is less receptive to lifestyle modifications or therapeutic additions. At this point, the physician is likely to be seeing the patient just for “a follow-up visit.”

This decline in cholesterol levels was emphasized in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries Trial (GUSTO). The GUSTO coordinating center reported that “post-MI patients who have high plasma cholesterol could be misclassified as being normocholesterolemic if they are tested at the wrong time.... To have a true estimate of the cholesterol level and to evaluate the individual risks correctly, blood specimens should be obtained within twenty-four hours of an AMI or at least three months following the infarction.”²² Unfortunately, the practice of waiting three months means a missed opportunity to educate the patient at a time when he or she is most receptive to adopting lifestyle changes (i.e., when recovering from an acute heart attack, bypass surgery, or angioplasty). Therefore, all Florida doctors—in fact all doctors—are urged to determine a patient’s lipid profile immediately upon suspicion of an acute cardiac event (i.e., when determining the presence or absence of enzymatic evidence of myocardial injury). If the cholesterol is elevated then, it is likely to be elevated in three months, and an active treatment program should be instituted. If the level is normal or even low, the patient should be challenged to adopt a heart-healthy lifestyle with the caveat that they must maintain these appropriate lifestyle habits. If at the three-month evaluation of the patient’s lipid profile the physician finds elevated cholesterol levels, a new and continuing therapy should be instituted. This compul-

siveness is indicated; the benefit of active treatment of the lipids has been repeatedly demonstrated.¹⁷⁻²²

Through regional and district meetings, efforts have been initiated to educate Floridians about the warning signs of AMI and the need for prompt attention in the ED. Through the Early Heart Attack Care program, Raymond Bahr, M.D., has led the way in redirecting attention to the earliest symptoms that may indicate an impending heart attack, and has placed emphasis on the importance of prodromal symptoms.²³ Dr. Bahr's enthusiasm, and that of his colleagues, has been—and continues to be—so great that it is unlikely that Florida can surpass Maryland in these efforts. But we will carefully study what Dr. Bahr recommends. In addition, new approaches are constantly sought to help patients with acute symptoms due to other vascular catastrophes.

It was recognized that much of the advice given to the public regarding prevention of a heart attack was similar, if not identical, to what the public should be taught to reduce their incidence of brain attack. In addition, a thrombolytic agent can frequently reduce and/or prevent disability (and even death) from brain attack if promptly administered in carefully selected patients who exhibit the earliest signs and symptoms. The U.S. Food and Drug Administration approved the use of tissue plasminogen activator (t-PA) for the treatment of non-hemorrhagic stroke in July 1996.²⁴ It was not surprising that EMSs were soon challenged to respond to patients experiencing the earliest signs of brain attack. It soon became clear that assistance and management for patients experiencing heart attack and those experiencing brain attack were similar in many respects; both are vascular disorders. A brain attack is a vascular disorder with neurologic consequences; a heart attack is a vascular disorder with unpredictable consequences. Therefore, those attending patients during the early minutes of a heart attack should be prepared to treat a brain attack as well.

One hundred fifty thousand Americans die each year because of brain attack; in 1995, 9,821 people died in Florida alone.²⁵ As many as three million Americans have survived brain attacks, but more than two million have permanent disabilities.

In 1966, the Florida Agency for Health Care Administration established a committee to develop guidelines for managing brain attacks.²⁶ It was clear that those trying to minimize the ravages of a brain attack would have to keep “an eye on the clock” just as those managing early treatment of AMI patients were urged to do. With advice and support from neurologists, neurosurgeons and neuroradiologists, the leaders of the FCACC decided that their program should be expanded to make Florida the safest state in which to have either a heart or brain attack.

Dr. Michael Gordon had already noted the importance of skilled recognition and emergency treatment of brain attack patients, and had already incorporated the idea into his EMS training program. Hopefully, in the not too distant future, all Florida EMS personnel will be skilled in the early emergent recognition and management of these patients.

But there are several caveats that must be emphasized when using thrombolytic agents to treat brain attack patients.^{24,26,27} First, the use of such an agent in patients who experience hemorrhagic stroke can be devastating. Of the 500,000 strokes sustained each year in this country, the majority (400,000) are ischemic in etiology. That leaves 100,000 that are hemorrhagic. The differences between hemorrhagic and ischemic stroke cannot usually be pinpointed with confidence when based only on patient history and physical examination; therefore, a quickly obtained and expertly interpreted computed tomography (CT) scan is essential prior to administering a thrombolytic agent. Patients with a heart attack will benefit most if the thrombolytic agent is administered within the first two hours after the onset of symptoms. The patient with the AMI will usually receive some benefit—and certainly no increased risk—if treatment is administered 12 or more hours after the onset of symptoms. However, the use of a thrombolytic agent more than three hours after the onset of a brain attack (even if ischemic in etiology) is potentially dangerous and can result in intracerebral hemorrhage. Therefore, as in heart attack treatment, the timing of treatment for brain attack is important, in fact, most important.

The difficulty lies in identifying the onset of the brain attack, and knowing when to “start the clock.” One-third of brain attacks occur while the patient is asleep. Patients frequently deny or neglect the early signs of a brain attack—signs such as vague numbness and weakness—and do not recall the time of onset.

Seldom has the balance between treatment, deliverance, and disaster been so finely poised. Clearly, each patient with a suspected brain attack must have a CT scan. This kind of requirement places a significant (if not insurmountable) burden on many hospitals.

In Florida, we are pleased with the progress that has been made in reducing the incidence of heart attack. We anticipate having a similar impact on the incidence and mortality of brain attack. Clearly the key to the success of this kind of program should be to reduce the incidence of vascular disease by primary and secondary prevention. Physicians need more enthusiasm and dedication to change their patients' adverse lifestyles. Many physicians may need to be “prodded” to become spokespersons for wellness. To this end, the FCACC joined with the American

Heart Association's Florida affiliate and encouraged the Governor of Florida (Mr. Lawton Chiles), to publicly proclaim April 1, 1997, as "the initiation of Florida's Campaign to Control Cholesterol." He urged "all citizens of the state of Florida to learn about cholesterol and blood pressure risk factors and have their levels checked regularly." Hopefully, this effort and the challenge given to the physicians of Florida by Dr. Bahr's contest will enlist more widespread action by physicians. But, is there a winner in Dr. Bahr's contest?

As more and more people survive heart and brain attacks—and postpone their first attack as well as the last "until later"—there has been a gratifying increase in our life expectancy. The life expectancy of an infant born when Social Security was established in 1935 was 62 years. Today, life expectancy is 72 years. In 1995, there were 55,000 citizens in this country who were 100 years or older; by the year 2030, there will be over one million people who fit that category.²⁹

It is likely that there are more centenarians living in Florida today than in Maryland. By 2030, the number will be even larger. It is unlikely that we will establish immortality in our state, or that Maryland physicians will establish it in theirs. What will people die of if they do not die of a heart or brain attack? Tuberculosis? Widespread metastatic cancer? AIDS or suicide? A heart attack for a centenarian who is still active may be better than one of these possibilities.

Thus, we are committed to working for the majority of our citizens dying young, late in life. We are committed to having more Floridians than Marylanders dying as young, vigorously active centenarians. To do this, we must prevent and/or successfully treat not only early occurring heart attacks, but brain attacks as well. In this sense, Florida will be the safest state in the union in which a physiologically young centenarian might have a heart or brain attack.

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Can Maryland become the first state to take heart disease out of first place where it has been since the turn of the century?

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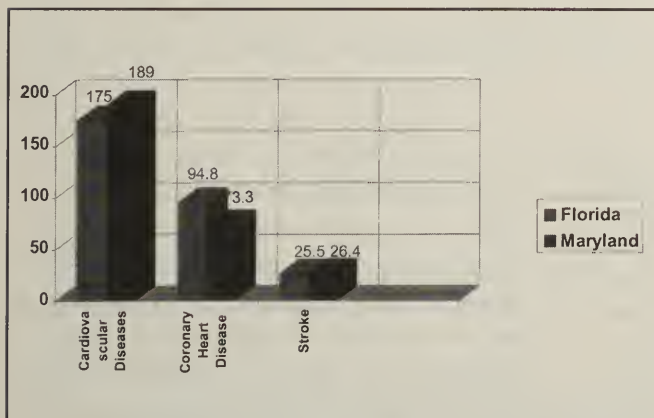


Figure 1. 1993 Age-Adjusted Death Rates (per 100,000 Population) for Total Cardiovascular Diseases, Coronary Heart Disease and Stroke between Florida and Maryland
1997 Heart and Stroke Statistical Update, American Heart Association

ABSTRACT: Heart disease has been the number one health problem in the United States since the turn of the century. It kills more Americans each year than all the American soldiers killed in all of this country's previous wars. Does it have to continue? Can this be reversed with re-engineering of information presently available? The hypothesis postulated here is that if a strategy can be successfully carried out in one state, then other states may be able to follow and pursue other strategies attempting to do the same. To accomplish this on a statewide level it is necessary to have individual hospitals proactively involved with their communities by having a game plan for penetration, identification, activation, and early management of patients with ischemic heart disease. Florida, under the leadership of Henry McIntosh, M.D., has attempted to put this into effect with a complicated strategy. Maryland attempts to logically put into place a chest pain strategy utilizing the principle of keeping it simple for widespread utilization.

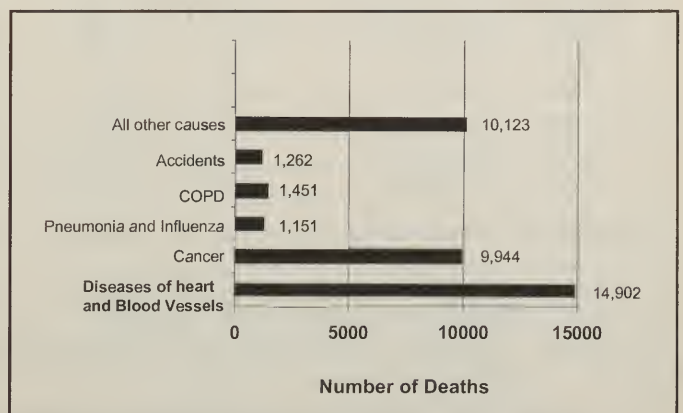


Figure 2. Leading Causes of Death in Maryland: 1992 Estimates

	<u>Florida</u>	<u>Maryland</u>
1. Counties	67	24
2. Population	14 million	5 million
3. Hospitals	292	83
4. Emergency Departments	260 approximately	58
5. Chest Pain Centers	50	25
6. Medical Schools	3	3
7. Deaths (per 100,000; 1993 data)		
Total Cardiovascular	174.9	73.3
Coronary Heart Disease	94.8	73.3
Stroke	25.5	26.4

<u>AGE OF CITIZENS</u>		
<u>Age</u>	<u>Florida</u>	<u>Maryland</u>
0-19	25%	27.6%
20-44	37%	41.7%
45-64	20%	19.6%
65+	18%	11.1%

Figure 3. Florida vs. Maryland Challenge.

1995 Population	County	Cardiovascular Deaths	Cancer Deaths	Re-education Needed
74,500	Allegany	439	294	146
459,700	Anne Arundel	1059	709	350
692,800	Baltimore City	3285	2200	1085
712,900	Baltimore County	2537	1699	838
64,000	Calvert	139	93	47
29,050	Caroline	124	83	41
138,800	Carroll	391	261	130
78,400	Cecil	252	168	84
111,300	Charles	206	138	69
30,350	Dorchester	183	122	61
175,000	Frederick	413	276	137
29,550	Garrett	121	81	41
209,100	Harford	489	327	161
218,400	Howard	303	203	100
18,750	Kent	97	65	33
810,000	Montgomery	1721	1153	569
771,600	Prince Georges	1539	1031	509
37,350	Queen Anne's	125	84	42
24,350	Somerset	132	88	44
80,900	St. Mary's	183	122	61
32,650	Talbot	159	106	53
127,400	Washington	477	319	158
79,400	Wicomico	321	215	107
39,350	Worcester	207	138	69
5,046,050	All Counties	14,902	9,944	4,959

Assumptions: 1997 data for counties reflects 1992 data
Diagnoses on death certificates are correct

Conclusions: A reduction of 4,959 cardiovascular deaths will take heart attack out of first place (our goal).

Figure 4. Maryland 1992 Heart Disease Statistics by County.

Can Maryland become the first state to take heart disease out of first place as a cause of death? Data on heart attack deaths within Maryland and Florida appear in **Figures 1, 2, and 3**. **Figure 4** outlines, by county, Maryland's population, current mortality rates from cardiovascular disease and cancer, and the reduction necessary to accomplish the goal. We start with a comparison by county because success must start somewhere. If one county can reduce heart attack deaths, it is only a matter of time before a single state, and then the nation, can do the same. For one county to achieve this goal, a community hospital must serve as the focus for community penetration and effective action. Only then can other hospitals be challenged to expand their capabilities to make Maryland the first state to take heart disease out of first place. And if Maryland can accomplish this, then so can other states.

Strategizing and engineering a plan to accomplish this goal needs to take into account evidenced-based medicine, but it also must generate a higher level of thinking that puts together the presently available elements needed for success. It is similar to continuous quality improvement, a process that brings together all of the players and states from the onset that the heart attack problem is a systems delivery failure and that identification of the problem can be sorted out. Identifying 20% of the major problems will impact 80% of the outcome. It is this type of strategy that we hope to employ in reducing heart attack deaths within the state of Maryland. To do so we need to identify the best practice to date that exists in chest pain centers (**Figure 5**). The chest pain strategy employs chest pain centers within hospital emergency departments as a way to penetrate communities. Each hospital serves as a community sarcomere for the heart attack problem. Simply stated, the chest pain center is based on identifying early patients with ischemic heart disease (prodromal) and putting together a plan for a comprehensive evaluation of such patients within the chest pain centers.

Some of the principles incorporated in this plan are as follows:

1. We first need to perfect the low probability ischemic disease pathway within the chest pain center (**Figure 6**).
2. We then have to educate the community about heart attack beginnings (prodromal symptom recognition).

- Focus on chest pain presentation as a way to get to the heart attack problem
- Establish a systematic approach: Comprehensive triage and management of chest pain patients
- Bring staff together. ACUTE CARDIAC TEAM (ACT)

Figure 5. Chest Pain Center Strategy.

**Perfecting the Low Probability Pathway Helps
Create a Vacuum That:**

- Shifts the paradigm to identify more prodromal patients.
- Encourages patients to act quickly and have a quick rule-out of their ischemia in the Chest Pain Center

Figure 6. Chest Pain Center Strategy.

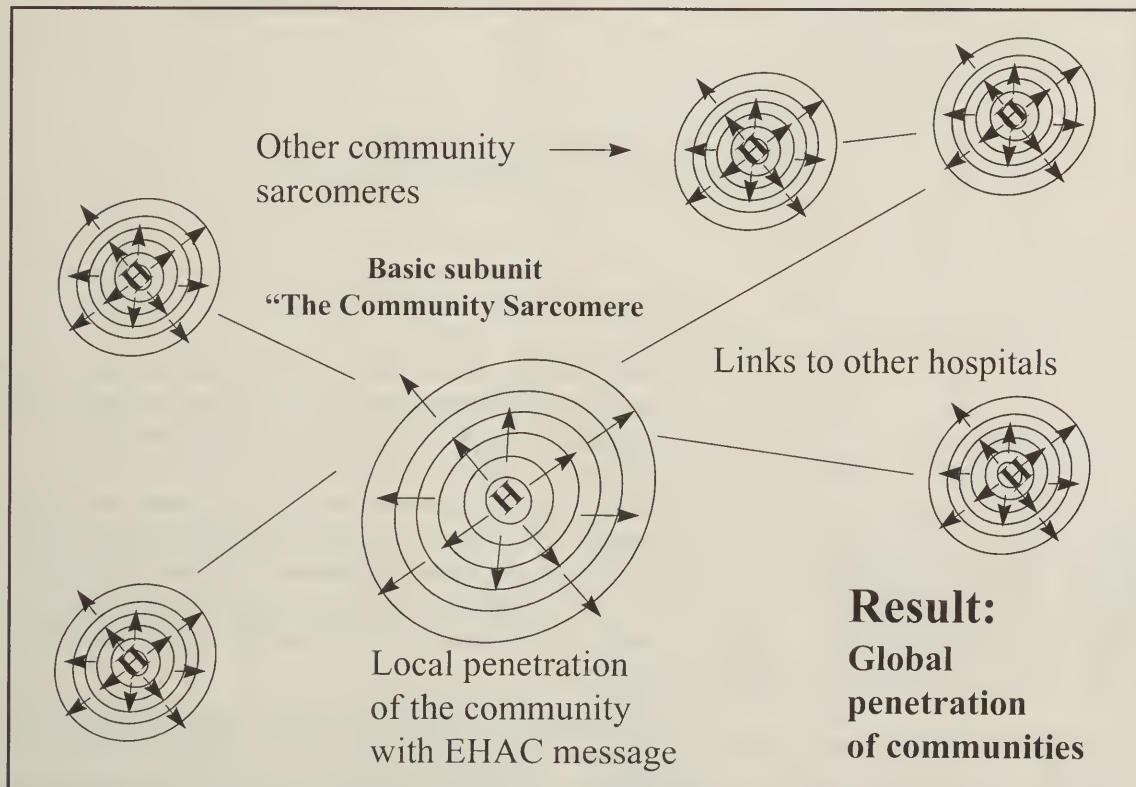


Figure 7. Reducing Heart Attack Deaths Through a Proactive Acute Prevention Program.

3. Finally, we have to link both of these elements with efficiency that enables optimal and maximal community penetration.

How do we get started, and how do we become part of the solution to the heart attack problem? We have to develop a plan of action.

1. The plan doesn't have to be perfect, but it has to work. When first discovered, electrocardiograms were based on the Einthoven triangle. In the early days, chapters in textbooks were written on how incorrect this assumption was, but the 12-lead electrocardiogram has become the standard and the best tool for identifying patients with acute myocardial infarction.
2. The plan has to best interpret the evidence we have at hand. Evidence-based medicine is very important, but Dr. Richard Horton, editor of *Lancet*, has pointed out that interpret-

ing best the facts that we have at hand becomes even more important as medicine is practiced frequently in the "gray" area between what we know and what we don't know.

3. The plan has to be simple or it will never work. The message in business for this is *KISS* (Keep It Simple Stupid).

In summary, what is needed for the heart attack problem is a simple plan that can spread and penetrate communities, linking together the elements and people in a cause that has great value. Using the early heart attack care message to bring people into the hospital earlier, and using chest pain units in emergency departments to be user-friendly in reception and comprehensive in management of such patients will become Maryland's strategy to address the heart attack problem on a statewide level. In doing so, Maryland encourages and challenges other states to likewise develop a strategy of their own to take heart disease out of first place within their state. (Figure 7). ■

Saint Joseph Medical Center emergency department chest pain center

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ABSTRACT: *Saint Joseph Medical Center has had two years experience with operating a chest pain center (CPC) in its emergency department. The CPC has resulted in improved treatment for patients with myocardial infarction. The CPC has led to the utilization of primary angioplasty as a preferred strategy for acute myocardial infarction. The CPC has allowed rapid rule-out of acute coronary syndromes in the emergency department thereby avoiding hospital admission in 31% of patients with chest pain of possible cardiac origin.*

The chest pain center (CPC) at Saint Joseph Medical Center (SJMC), Baltimore, Maryland, commenced operations in May 1995 with the dual purpose of providing more rapid and effective treatment for patients with acute myocardial infarctions, and providing a cost-effective and efficient way of evaluating chest pain and ruling out acute coronary syndromes. To date, approximately 6,000 patients with a chief complaint of chest pain have been evaluated. Two thousand patients were felt to have clearly noncardiac chest pain after initial physician evaluation and electrocardiogram (ECG), and did not require further cardiac evaluation. Three thousand nine hundred and nineteen patients were felt to have chest pain of possible cardiac etiology requiring further evaluation. The average door to ECG time for all chest pain patients was seven minutes. The door to ECG time for patients who had transmural myocardial infarctions (MIs) was four minutes.

Of the 3,919 patients with possible cardiac chest pain, 56% (2,195) were felt to be at high risk for an unstable coronary syndrome (acute myocardial infarction or unstable angina). These patients were admitted to the hospital after initial emergency department (ED) evaluation and stabilization, but did not go through the chest pain evaluation unit (CPEU). Twenty-nine percent were admitted to the critical care unit or intensive care unit; 71% were admitted to an intermediate level unit with cardiac telemetry.

Forty-four percent (1,724) of the patients with possible cardiac chest pain who were felt to be at moderate or low risk were evaluated in the

emergency department's CPEU. Of these CPEU patients, 72% (1,233) were discharged to home from the emergency department, and 28% (475) were admitted to the hospital. The average length of stay for the CPEU patient was 6.5 hours. Thirty-four percent of these patients had treadmill stress tests, and 4% had nuclear scans.

The CPEU protocol involves initial physician evaluation and ECG followed by a risk stratification using the guidelines for unstable angina promulgated by the Agency for Health Care Policy and Research. Patients at high risk are admitted to the hospital; patients at low to moderate risk are placed in the CPEU. The only exceptions are patients who may be at low risk, but who for one reason or another would not be able to undergo the necessary testing (including stress tests) contained in the CPEU protocol.

The rule-out protocol involves continuous ST-segment monitoring, and three sets of hourly ECGs and blood studies. Each of the three sets of blood tests include CPK, MB, and cardiac troponin I. In addition, serum myoglobin is assayed on the first two sets of blood. If a patient waits overnight for a stress test, a troponin is repeated in the morning before the test. Patients with ongoing pain may have a resting sestimibi cardiac perfusion scan. Patients who have been symptom-free typically are given a Bruce Protocol stress test. A small number of patients are given a stress test with nuclear imaging or a non-treadmill exercise stress test. Some patients who are felt to be at very low risk may be discharged and brought back for stress testing in the morning. Some patients are referred to their cardiologist for stress tests within 72 hours after appropriate consultation.

Stress tests are done from 8:00 a.m. to 6:00 p.m. seven days a week. The stress tests are performed by staff cardiologists from the department of medicine, division of cardiology, SJMC. Of the 450 stress tests ordered by the emergency department physicians, no significant complications have arisen. The test has been highly reliable with only one false negative — a result that, in retrospect, seemed to be interpreter error.

During the last year, a 12-lead telemetric ECG from the field has been available through cooperation with the Baltimore County Fire Department. In patients with acute MIs these ECGs are of great benefit in preparing for their ED arrival. On several occasions, preparation for thrombolysis was completed by the time the patient arrived. In other circumstances, patients have gone directly to the catheterization laboratory from the ambulance without experiencing any delay in the emergency department.

In mid-1996, SJMC started using immediate percutaneous transluminal coronary angioplasty (PTCA) without prior thrombolytics (primary PTCA) as the preferred treatment for acute MI. However, PTCA is only available 13 hours per day, from 7:00 a.m. to 8:00 p.m. The same National Heart Attack Alert Program guidelines that we use for thrombolysis (i.e.,

door to ECG in five minutes; door to treatment in 30 minutes) are applied to PTCA.

We find that usually patients can be in the catheterization laboratory just as quickly as the emergency department can administer thrombolytics. The experience of this institution has been that the time to reperfusion from the onset of treatment is much less for patients with angioplasty than with thrombolysis. (It should be noted that SJMC has four catheterization laboratories, two of which are immediately adjacent to the emergency department.)

Since April 1996, 112 patients have been taken directly from the ED to the catheterization laboratory. Of these, 53 were sent for MI. The others were sent for presumed unstable angina. Of the MI patients, the majority of these were for primary angioplasty, but some required the catheterization laboratory for rescue angioplasty (i.e., angioplasty required when thrombolytics fail to produce reperfusion).

The emergency physicians and consulting cardiologists of SJMC are convinced that primary angioplasty is superior to thrombolytics during the hours when the catheterization laboratory is open. These patients do very well and have few complications. Their length of stay is often two to three days, sometimes with no residual evidence of infarction. Furthermore, this process proves a greater cost savings than the use of more traditional therapies. Formally collected data is being evaluated to objectively assess time versus treatment, outcomes, and costs. Hopefully this data will support an extension of catheterization laboratory availability. Although there is an emergency on-call catheterization team when the catheterization laboratory is closed, the time constraints of primary PTCA requires the team to be on-site to be a preferred strategy to thrombolysis.

Overall, there is a consensus of the SJMC physicians and staff that the chest pain center has led to improved treatment for patients with chest pain. Ambulance patients with acute MIs can have treatment prepared before arrival by use of prehospital ECG transmission. Definitive therapy with thrombolytics can now be administered in 30 minutes. Primary angioplasty with comparably short door-to-wire times has become a viable strategy. Patients with nontransmural MIs are quickly identified while other patients can safely and effectively be "ruled-out" in hours rather than days. In conclusion, the CPC has focused the entire staff on optimizing care to the chest pain patient.

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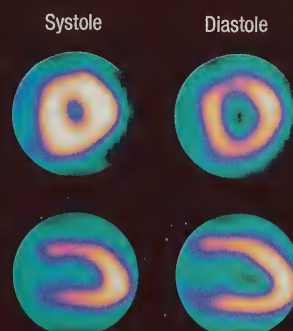
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There have been infrequent reports of signs and symptoms consistent with seizure and severe hypersensitivity after administration of Tc99m Sestamibi. Please see brief summary of prescribing information on adjacent page.

Cardiolite®

Kit for the preparation of Technetium Tc99m Sestamibi

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Brief Summary

Cardiolite®

Kit for the preparation of Technetium Tc99m Sestamibi

FOR DIAGNOSTIC USE

INDICATIONS AND USAGE: CARDIOLITE®, Kit for the preparation of Technetium Tc99m Sestamibi, is a myocardial perfusion agent that is indicated for detecting coronary artery disease by localizing myocardial ischemia (reversible defects) and infarction (non-reversible defects), in evaluating myocardial function and developing information for use in patient management decisions. CARDIOLITE® evaluation of myocardial ischemia can be accomplished with rest and cardiovascular stress techniques (e.g., exercise or pharmacologic stress in accordance with the pharmacologic stress agent's labeling).

It is usually not possible to determine the age of a myocardial infarction or to differentiate a recent myocardial infarction from ischemia.

CONTRAINDICATIONS: None known.

WARNINGS: In studying patients in whom cardiac disease is known or suspected, care should be taken to assure continuous monitoring and treatment in accordance with safe, accepted clinical procedure. Infrequently, death has occurred 4 to 24 hours after Tc99m Sestamibi use and is usually associated with exercise stress testing (See PRECAUTIONS).

Pharmacologic induction of cardiovascular stress may be associated with serious adverse events such as myocardial infarction, arrhythmias, hypotension, bronchoconstriction and cerebrovascular events. Caution should be used when pharmacologic stress is selected as an alternative to exercise; it should be used when indicated and in accordance with the pharmacologic stress agent's labeling.

PRECAUTIONS:

GENERAL

The contents of the vial are intended only for use in the preparation of Technetium Tc99m Sestamibi and are not to be administered directly to the patient without first undergoing the preparative procedure.

Radioactive drugs must be handled with care and appropriate safety measures should be used to minimize radiation exposure to clinical personnel. Also, care should be taken to minimize radiation exposure to the patients consistent with proper patient management.

Contents of the kit before preparation are not radioactive. However, after the Sodium Pertechnetate Tc99m Injection is added, adequate shielding of the final preparation must be maintained.

The components of the kit are sterile and non-pyrogenic. It is essential to follow directions carefully and to adhere to strict aseptic procedures during preparation.

Technetium Tc99m labeling reactions involved depend on maintaining the stannous ion in the reduced state. Hence, Sodium Pertechnetate Tc99m Injection containing oxidants should not be used.

Technetium Tc99m Sestamibi should not be used more than six hours after preparation.

Radiopharmaceuticals should be used only by physicians who are qualified by training and experience in the safe use and handling of radionuclides and whose experience and training have been approved by the appropriate government agency authorized to license the use of radionuclides.

Stress testing should be performed only under the supervision of a qualified physician and in a laboratory equipped with appropriate resuscitation and support apparatus.

The most frequent exercise stress test endpoints, which resulted in termination of the test during controlled Tc99m Sestamibi studies (two-thirds were cardiac patients) were:

Fatigue	35%
Dyspnea	17%
Chest Pain	16%
ST-depression	7%
Arrhythmia	1%

Carcinogenesis, Mutagenesis, Impairment of Fertility

In comparison with most other diagnostic technetium labeled radiopharmaceuticals, the radiation dose to the ovaries (1.5rads/30mCi at rest, 1.2 rads/30mCi at exercise) is high. Minimal exposure (ALARA) is necessary in women of childbearing capability. (See Dosimetry subsection in DOSAGE AND ADMINISTRATION section.)

The active intermediate, [Cu(MIBI)₄]BF₄, was evaluated for genotoxic potential in a battery of five tests. No genotoxic activity was observed in the Ames, CHO/HPRT and sister chromatid exchange tests (all *in vitro*). At cytotoxic concentrations ($\geq 20\mu\text{g/ml}$), an increase in cells with chromosome aberrations was observed in the *in vitro* human lymphocyte assay. [Cu(MIBI)₄]BF₄ did not show genotoxic effects in the *in vivo* mouse micronucleus test at a dose which caused systemic and bone marrow toxicity (9mg/kg, $> 600 \times$ maximal human dose).

Pregnancy Category C

Animal reproduction and teratogenicity studies have not been conducted with Technetium Tc99m Sestamibi. It is also not known whether Technetium Tc99m Sestamibi can cause fetal harm when administered to a pregnant woman or can affect reproductive capacity. There have been no studies in pregnant women. Technetium Tc99m Sestamibi should be given to a pregnant woman only if clearly needed.

Nursing Mothers

Technetium Tc99m Pertechnetate is excreted in human milk during lactation. It is not known whether Technetium Tc99m Sestamibi is excreted in human milk. Therefore, formula feedings should be substituted for breast feedings.

Pediatric Use

Safety and effectiveness in children below the age of 18 have not been established.

ADVERSE REACTIONS: During clinical trials, approximately 8% of patients experienced a transient parosmia and/or taste perversion (metallic or bitter taste) immediately after the injection of Technetium Tc99m Sestamibi. A few cases of transient headache, flushing, edema, injection site inflammation, dyspepsia, nausea, vomiting, pruritus, rash, urticaria, dry mouth, fever, dizziness, fatigue, dyspnea, and hypotension also have been attributed to administration of the agent. Cases of angina, chest pain, and death have occurred (see WARNINGS and PRECAUTIONS). The following adverse reactions have been rarely reported: signs and symptoms consistent with seizure occurring shortly after administration of the agent; transient arthritis in a wrist joint; and severe hypersensitivity, which was characterized by dyspnea, hypotension, bradycardia, asthenia and vomiting within two hours after a second injection of Technetium Tc99m Sestamibi.

DOSAGE AND ADMINISTRATION: The suggested dose range for I.V. administration in a single dose to be employed in the average patient (70kg) is:

370-1110MBq (10-30mCi)

The dose administered should be the lowest required to provide an adequate study consistent with ALARA principles (see also PRECAUTIONS).

When used in the diagnosis of myocardial infarction, imaging should be completed within four hours after administration (see also CLINICAL PHARMACOLOGY).

The patient dose should be measured by a suitable radioactivity calibration system immediately prior to patient administration. Radiochemical purity should be checked prior to patient administration. Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration whenever solution and container permit.

Store at 15-25°C before and after reconstitution.

RADIATION DOSIMETRY: The radiation doses to organs and tissues of an average patient (70kg) per 1110MBq (30mCi) of Technetium Tc99m Sestamibi injected intravenously are shown in Table 4.

Table 4. Radiation Absorbed Doses from Tc99m Sestamibi

Organ	Estimated Radiation Absorbed Dose			
	REST			
	2.0 hour void		4.8 hour void	
	rads/ 30mCi	mGy/ 1110MBq	rads/ 30mCi	mGy/ 1110MBq
Breasts	0.2	2.0	0.2	1.9
Gallbladder Wall	2.0	20.0	2.0	20.0
Small Intestine	3.0	30.0	3.0	30.0
Upper Large Intestine Wall	5.4	55.5	5.4	55.5
Lower Large Intestine Wall	3.9	40.0	4.2	41.1
Stomach Wall	0.6	6.1	0.6	5.8
Heart Wall	0.5	5.1	0.5	4.9
Kidneys	2.0	20.0	2.0	20.0
Liver	0.6	5.8	0.6	5.7
Lungs	0.3	2.8	0.3	2.7
Bone Surfaces	0.7	6.8	0.7	6.4
Thyroid	0.7	7.0	0.7	6.8
Ovaries	1.5	15.5	1.6	15.5
Testes	0.3	3.4	0.4	3.9
Red Marrow	0.5	5.1	0.5	5.0
Urinary Bladder Wall	2.0	20.0	4.2	41.1
Total Body	0.5	4.8	0.5	4.8

Organ	STRESS			
	2.0 hour void		4.8 hour void	
	rads/ 30mCi	mGy/ 1110MBq	rads/ 30mCi	mGy/ 1110MBq
Breasts	0.2	2.0	0.2	1.8
Gallbladder Wall	2.8	28.9	2.8	27.8
Small Intestine	2.4	24.4	2.4	24.4
Upper Large Intestine Wall	4.5	44.4	4.5	44.4
Lower Large Intestine Wall	3.3	32.2	3.3	32.2
Stomach Wall	0.5	5.3	0.5	5.2
Heart Wall	0.5	5.6	0.5	5.3
Kidneys	1.7	16.7	1.7	16.7
Liver	0.4	4.2	0.4	4.1
Lungs	0.3	2.6	0.2	2.4
Bone Surfaces	0.6	6.2	0.6	6.0
Thyroid	0.3	2.7	0.2	2.4
Ovaries	1.2	12.2	1.3	13.3
Testes	0.3	3.1	0.3	3.4
Red Marrow	0.5	4.6	0.5	4.4
Urinary Bladder Wall	1.5	15.5	3.0	30.0
Total Body	0.4	4.2	0.4	4.2

Radiopharmaceutical Internal Dose Information Center, July, 1990, Oak Ridge Associated Universities, P.O. Box 117, Oak Ridge, TN 37831, (615) 576-3449.

HOW SUPPLIED: Du Pont Radiopharmaceutical's CARDIOLITE®, Kit for the Preparation of Technetium Tc99m Sestamibi is supplied as a 5ml vial in kits of two (2), five (5) and thirty (30) vials, sterile and non-pyrogenic.

Prior to lyophilization the pH is between 5.3-5.9. The contents of the vials are lyophilized and stored under nitrogen. Store at 15-25°C before and after reconstitution. Technetium Tc99m Sestamibi contains no preservatives. Included in each two (2) vial kit are one (1) package insert, six (6) vial shield labels and six (6) radiation warning labels. Included in each five (5) vial kit are one (1) package insert, six (6) vial shield labels and six (6) radiation warning labels. Included in each thirty (30) vial kit are one (1) package insert, thirty (30) vial shield labels and thirty (30) radiation warning labels.

The U.S. Nuclear Regulatory Commission has approved this reagent kit for distribution to persons licensed to use byproduct material pursuant to section 35.11 and section 35.200 of Title 10 CFR Part 35, to persons who hold an equivalent license issued by an Agreement State, and, outside the United States, to persons authorized by the appropriate authority.



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On lifting the fog of war in the battle on heart disease: Star Wars technology in pursuit of a seamless integration strategy

James A. Espinosa, M.D., F.A.C.E.P., F.A.A.F.P. (Part I and III), Linda K. Kosnik, RN, CEN (Part I and III), Michael Kraitsik (Part II), and Jeffrey C. Dillow (Part III)

ABSTRACT: *In our efforts to reduce cardiac morbidity and mortality we often use terms such as the “battle” or “war” on heart disease. If we believe efforts to reduce cardiac disease are the moral equivalent of war, then perhaps we should explore ways that military strategic and tactical metaphors can be applied through technology to the cardiac battle. In this article we explore three major areas for technological advancement: adaptation of the strategies of outcomes management and evidence-based medicine, computer simulation and animation efforts to create horizontal and vertical integration of strategic efforts, and use of interactive multimedia in “recruiting an army” through community empowerment. The overall goal is to find ways to lift “the fog of war” in the battle on heart disease, in order to further the integration of our various efforts.*

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PART I

Health care requires a central nervous system—the new technology of the science of outcomes management

Introduction

Carl von Clausewitz was born in 1780 in what was recently, until 1990, East Germany. He entered the Prussian army in 1792 and was admitted to the War College in Berlin in 1801. He served with both the Russian and Prussian armies in the Napoleonic wars. In 1818, he was made the head of the War College. In the next twelve years until his death, he wrote a number of brilliant works on war theory. He is known for many critical concepts about war tactics and strategy. Perhaps his most famous metaphor is the “Fog of War.” This notion suggests that military strategy is made more complicated by difficulties in seeing the battlefield. Military commanders in Desert Storm found this concept to be true; many deaths were attributed to “friendly fire.”

The U.S. military has made consistent efforts to find technological solutions to the “Fog of War.” Some of these solutions involve multimedia and battlefield animation. If we believe efforts to reduce deaths from heart disease are the moral

equivalent of war, then perhaps we should consider some of these technological breakthroughs.

The goal of a seamless integration of the chest pain center with other chest pain treatment strategies is difficult. Multiple practitioners are involved. The goal of horizontal and vertical integration of health care is a lofty ideal whose practical cognizance is far from realized. Current information systems do not generally provide the data needed to make real-time decisions. Integration of new research into practice is challenging. Most importantly, the patient is a combatant in the war on heart disease.

We discuss three major areas for technology advancement. The first is an appreciation of the new intellectual technology of the outcomes movement and evidence-based medicine. As Ellwood has so elegantly framed the problem, "The intricate machinery of our health care systems can no longer grasp the threads of experience...too often payers, physicians, and health care executives do not share common insights into the lives of the patients. We acknowledge that our common interest is the patient, but we represent this interest from such divergent, even conflicting viewpoints that everyone loses perspective." As a result, Ellwood sees that "health care is unstable, confused and...in need of a central nervous system."

The second approach involves computer simulations and animations in our tactical and strategic work. The last is the use of interactive multimedia, in the context of the problem of early heart attack care (EHAC) and cardiac awareness.

It is hoped that these new directions will spur interest in these tools as adjuncts to the Maryland chest pain center research movement.

The new intellectual technology of the outcomes movement: a perspective from emergency medicine, applicable to entire house of medicine

An American College of Emergency Physicians (ACEP) Statement of Direction, dated April 6, 1995, identified a number of goals and objectives for the College. It was specifically noted under the Profession Practice goals that "ACEP will insure that outcome measurement systems will be available as a tool to enable members to guide their emergency medicine practice." However, this theme was supported by objectives itemized under other goals. Under Advocacy, an objective was crafted that promoted emergency medicine as an essential element of the health care delivery system, and that based on research ACEP will promote improvements in our practice environment. Several of the research objectives could be supported by a better understanding of the outcomes movement. For example, it is stated that ACEP will promote the most effective and efficient emergency medicine patient care methodologies, will acquire on the emergency medicine practice environment, and will "research and teach members what clinical practice data are needed, how to obtain it, and to clearly present it in support of their own product."

A recent Research Directions Conference of ACEP and Society of Academic Emergency Medicine developed a research

agenda for emergency medicine. The consensus document specifically called for the development of new methods to assess the outcomes of emergency care. "New research methods are needed to assess health care outcomes, quality of care and costs in terms of human suffering, anxiety, quality of life, and disability. It will take time and effort to identify and apply valid and reproducible measures of pain, anxiety, quality of life, functional status, disability, and satisfaction with emergency care. Better measures of emergency care costs, cost-effectiveness, cost-benefit are also needed. Assessment of health care cost must take into account the ability of the population to access the existing health care system and cost of delays in health care."

This notion of understanding and adapting to a shifting health care environment has been a consistent theme for many emergency medicine thought leaders. For example, Dr. Greg Henry has minced no words concerning his conclusions about how third-party payers will evaluate emergency physicians. According to Henry, emergency physicians will be judged on the following attributes: cost-effectiveness, malpractice, maintenance of business, and adherence to standards.

Furthermore, Henry has challenged us to "focus on why the College exists." He sees the need to direct College energies in four directions: productivity resource utilization ("evidence-based medicine will rule"), cost versus charge, value-added services need to be explored, and central hub theory of medical care must predominate. One can readily see how these concepts apply to the problem of the chest pain center.

A brief history of the development of a chest pain center/observation unit at Overlook Hospital

Overlook Hospital in Summit, New Jersey, opened a chest pain center on February 14, 1994. The goals of the chest pain center are to optimize time to treatment, provide cost-effective treatment for low-risk chest pain patients, and provide an appropriate level of care to each patient.

A task force was formed in 1993 with the purpose of creating a chest pain observation unit. The members identified Raymond Bahr, M.D., as national leader in the development of chest pain centers. The local champion was identified as Dr. William Tansey, III, M.D. Dr. Tansey is a cardiologist at Overlook Hospital. He was instrumental in bringing the team together and in supporting its ongoing work.

The team identified a need for a dedicated physical space, as well as for enhanced laboratory and cardiology services. The team also felt that the relationship between cardiologists, emergency nurses, and emergency physicians should be enhanced. Informatic support was needed, as well as some monetary resources to create, staff, and manage the center.

The driving forces toward the creation of the center were a commitment to state-of-the-art care, as well as an awareness of the need to respond to emerging capitated managed care contracts. Other driving forces included existing expertise in total quality management/continuous quality improvement (TQM/

CQI) applications, as well as expertise in data analysis and presentation.

Baseline data were acquired, including the number of chest pain patients seen, and the length of stay for low-risk chest pain patients who were admitted to the hospital. Estimates were made of the future daily patient load of chest pain patients who might be eligible for a chest pain center, and some determinations of unit size were performed. The team determined that a five-bed chest pain center was an initial goal, with a minimum of a two-bed center.

The team felt that an ideal center would be near the main emergency department (ED), would be quiet and comfortable, and would be monitored. Protocols were developed and approved by cardiologists, emergency physicians, and nurses. These protocols described admission criteria to the center, as well as standing orders for the management of chest pain patients.

The center's volume has increased steadily since its inception. The multidisciplinary Chest Pain Performance Improvement Team meets regularly to monitor the chest pain center. Future directions of the center include the development of a patient satisfaction survey. The team constantly looks to create improvements to the protocols which manage the center. New physical plant improvements are in process. The ongoing literature concerning advances in methods for early detection is a standing agenda item.

PART II

Seeing the battlefield, visualizing tactics and strategies: the application of computer simulation technologies

The Fog of War is a real and significant challenge that we must face in the battle to improve cardiac outcomes. This "fog" creates an environment in which it is difficult to visualize the integration of overlapping processes, such as the prehospital system, emergency department, ancillary services, and in-patient and out-patient services.

Historically, the organization of health care systems has resulted from a lack of a shared vision. Health care managers are confronted with crucial decisions, including, management of their operations, increased competition, fixed-rate reimbursement, and declining hospital occupancy. Therefore, the management of capacity (work force, equipment, and facilities) becomes of paramount importance in order to maintain a competitive edge. This is both a *strategic issue*, in which the system needs to be viewed from a high level (the so-called 10,000 foot level), as well as a *tactical issue*, in which the individual departments need to be optimized in light of the whole, necessitating a low-level look (the so-called 100 foot level).

Computer simulation is a tool that can provide the necessary robustness to analyze these problems. Computer simulation has been a tool widely applied, in the manufacturing environment, to problems of capacity analysis, equipment planning and scheduling, bottleneck identification, turnaround time analysis, regional services planning, and facilities design. More recently with the advent of animated, user-friendly software packages,

this tool has been applied to more non-traditional environments such as health care. Computer simulation modeling creates a virtual world in which the health care system becomes alive using the latest in animated computer tools. It actually shows a selected health care system as it behaves over time, with all of its operational and probabilistic events on the screen. Computer simulation models are not only visually correct but also logically and mathematically correct in their ability to do prediction with "what-if" scenarios.

Computer simulation integrates several key quality improvement techniques, and can be used in strategic and tactical applications.

Example of a strategic application: regional health care planning for cardiac services

A project is currently underway in Monroe County (Rochester), New York. Seven hospitals and their related emergency medical services (EMS) services are involved in the project. A simulation model is being used to optimize the service delivery system. This model takes the strategic view of 10,000 feet. The animation takes place on a map of the Monroe County area, and shows ambulances and helicopters transporting patients with chest pain. The model then simulates the progress of the patient from admission to discharge. The model will look at ways to shorten the onset of chest pain to definitive treatment.

Example of a tactical application: simulation applied to the design of a new emergency department

Simulation modeling aids the design of a new emergency department. This model takes the tactical view of 100 feet. The animation takes place on a layout of the emergency department. The model evaluates alternative layouts including: number of rooms, equipment requirements, staffing levels, requirements of ancillary services, and the effect of these elements on costs, length of stay, and quality of care.

PART III

Recruitment of the Army, Star Wars level visualization of the EHAC message: the use of interactive multimedia techniques. The first phase of the Community Schoolhouse Project.

The Overlook Hospital ED has a commitment to improving the management of the patient with chest pain. We have tied that commitment to an interest in health care applications of the industrial model of continuous process improvement and total quality management.

Our research in this area to date is perhaps best summarized by a review of an article in the recently published proceedings of the Second National Congress of Chest Pain Centers in Emergency Departments. This article reviews the journey of a quality improvement team, which challenged itself to create a chest pain center as well as to reduce time to thrombolytic treatment in patients with myocardial infarction. The team was successful on

both accounts. Time to thrombolytic treatment was reduced in a dramatic, sustained, and clinically significant way. This work has been recognized as a HCFA Best Practice Benchmark Project. The chest pain center grew from a two-bed, intra-ED designated bed unit into a two-bed dedicated unit, and now into a four-bed dedicated chest pain unit.

However, as the article notes, our work in reducing time to thrombolytic treatment in the emergency setting, and in developing a chest pain center, has led us into an ever more challenging and important direction. It has given us cause to think about creative ways to apply our CQI/TQM experience to the *prehospital* arena.

As we all know, many patients wait hours before accessing the health care system, often with tragic consequences.

We have asked ourselves how to apply a TQM/CQI approach towards reducing time to ED *presentation* in patients with chest pain, on a *community level* of analysis and intervention.

Possible restraining forces to community-level interventions to reduce time to thrombolytic treatment

Some have expressed concerns that there are serious gaps in the current science base on the issue of community-level interventions of this sort. The National Institutes of Health, in fact, has designed a controlled trial, called the Rapid Early Action for Coronary Treatment trial. After such a trial, we might be better equipped to implement educational and other strategies in an efficient and cost-effective manner.

The case for community-level interventions to reduce time to thrombolytic treatment

On the other hand, precious lives may be lost waiting for randomized trial data. One might even hypothesize that even if there were no significant differences between intervention and nonintervention communities in a randomized trial, it would not necessarily imply that all such efforts are fruitless. The TQM philosophy would suggest that we might consider enhancements and changes to the nature of the interventions, with subsequent restudy. Additionally, one might reflect on the national experience with smoking cessation intervention, in which meaningful changes in smoking behavior lagged decades behind initial community education campaigns.

Current knowledge concerning intervention programs to reduce patient delays in acute myocardial infarction is well reviewed in the previously cited clinician document. Johan Herlitz, M.D., reviewed the impact of educational campaigns on patient delay to presentation. Gordon A. Ewy, M.D., also discussed causes of patient delay in presenting with acute myocardial infarction. A telling point in his article concerns his opinion that, "Patient education aimed at

altering patient behavior after onset symptoms has not been encouraging."

We have reviewed the literature on reducing prehospital patient delay and believe that our TQM/CQI experience is informative in helping us to conceive an approach that would be successful. Our efforts to date have centered around the concept which we have called the "EMS Bridge to the Community."

Our work to date has been in learning to utilize the EMS system as a bridge to the community. EMS personnel, as natural ambassadors to the community, represent a bridge from the ED to the community, and from the community to the ED. The EMS personnel can be seen as persons with high profile and credibility in the communities in which they work and live. Our work to date in utilizing this concept has involved increased training of EMS personnel, at all levels, in the core concepts of EHAC, the open-artery hypothesis, and the immense value of a prehospital 12-lead electrocardiogram. It has been a building block to community-oriented TQM-driven interventions.

The "EMS bridge" was a step in the road to satisfy our need to do more, and to reduce time to prehospital treatment, while we create the infrastructure needed for meaningful and lasting behavioral change in the community.

The heart of this project is the "Community Schoolhouse Bridge/Virtual EHAC Center"

Project Objectives:

- Explore a new dimension in patient education technology known as interactive multimedia.

We are in the process of the development of an interactive program designed to powerfully and robustly communicate the EHAC message. The core of this approach is the creation of a virtual "EHAC Schoolhouse." This virtual facility is a interactive multimedia environment in which users can interact with EHAC concepts messages and lessons in an easy and nonthreatening manner.

The target audience includes children as well as adults of all ages. There are thirteen proposed "areas" in the total "site." These "sites" each feature specific interactivity, with specialized content material to convey. Core messages are reinforced throughout the entire program. Users can tour a virtual chest pain center and can become desensitized to the testing procedures. The goal is to reinforce EHAC messages while exposing the user to the wonders of cardiac physiology. We are trying to create a robust antithesis to messages of fear, which trigger denial.

- Enhance patient participation and responsibility.

We will provide feedback to the community concerning time to presentation with chest pain, so that a sense of ownership of the problem can be enhanced. Educational programs in the Schoolhouse will include this concept.

- Develop mentorship between senior citizens, adults, adolescents, and children in the community.

EHAC tools are aimed at a spectrum of age groups. We will work to coordinate some of the educational efforts of EHAC by developing mentorship between generations.

- Coordinate educational efforts with those of the private physicians in the office setting, and with managed care providers.

Dr. Ewy pointed out in his lecture, reviewed in *Clinician*, that “involvement of the patient’s primary care physician delays patient therapy. This is of special concern in this era of managed care.”

We will attempt to demonstrate that the needs of managed care providers, and of private physicians, can be aligned through preventive education.

- Expand on our existing database of time to presentation of patients with chest pain. Better understand the role of the “stuttering chest pain presentation.”

We have a database that gives us some insight into the problem of the prehospital interval (time to patient presentation to the ED.)

We would expand on this database through the development of better ways of identifying patients with stuttering chest pain presentations. Our current data suggest that we have a bimodal distribution of time to presentation of patients with myocardial infarction. Approximately half of our patients have a *delayed* time to presentation, consistent with the notion of a “stuttering chest pain presentation.”

We need to thoroughly understand on a prospective basis how best to interview patients concerning their time of onset of symptoms.

Conclusion

If we believe the notion of a battle in the war on heart disease is more than a metaphor, we ought to be prepared to think strategically and tactically. In this article we have discussed three technologies, all designed to support and meet the EHAC challenge and reduce the current “Fog of War.”

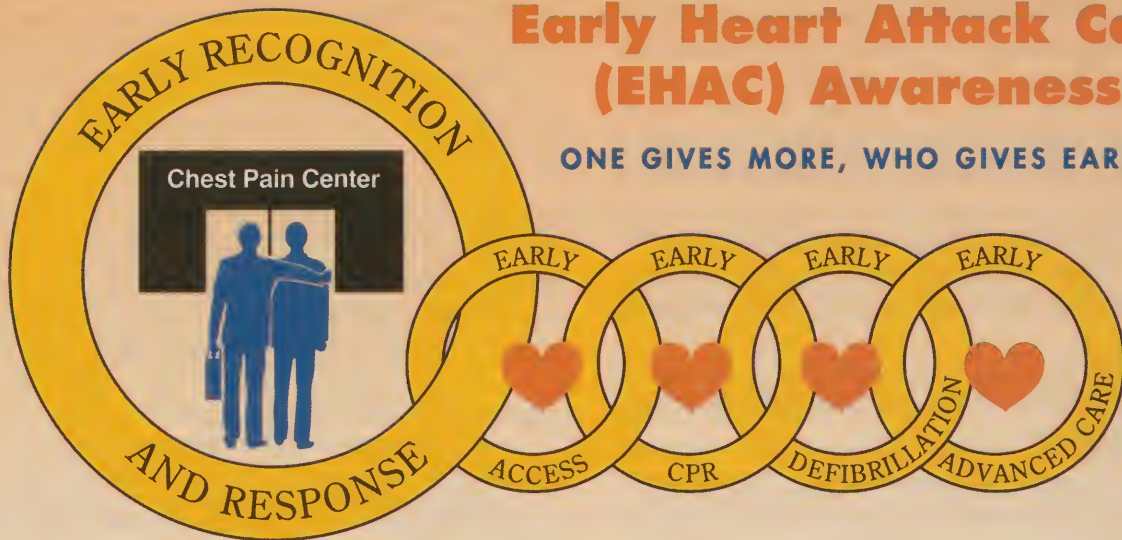
We propose that outcomes management is more than just an intellectual exercise. It is a powerful technology in and of itself. We propose that computer simulation can be used to intelligently deploy our resources. Lastly, we have described a project in which we are designing an interactive, multimedia “virtual EHAC center” with the goal of recruiting an ever-growing army in the battle on heart disease.

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Early Heart Attack Care (EHAC) Awareness

ONE GIVES MORE, WHO GIVES EARLY



Chest Pain Centers: Management of Central Chest Pain

A Chest Pain Center (CPC) is an expansion of the hospital's heart station into the emergency department (ED). A Chest Pain Center (CPC) serves as a command post for quickly identifying and attending to patients experiencing or at risk to experience a heart attack.

In compliance with ACEP Guidelines, a CPC includes these seven features and functions:

- 1** Fast track for patients with acute myocardial infarction.
- 2** Observation area for patients with acute myocardial ischemia.
- 3** Detection program to target and educate patients with coronary artery disease (based on a positive stress test) and patients with increased risk factors (based on history and blood tests).
- 4** Education outreach program in conjunction with the hospital's rehabilitation department, to communicate to the public that warning signs precede heart attacks.
- 5** Medical educational programs involving CQI, HCFA MI indicators, NHAAP recommendations, etc. The goal is the relentless pursuit of teamwork for maximal benefit to the patient, the hospital, and society.
- 6** Proper staffing ratio of critical care nurses, as well as critical care physicians with a continuous retraining program for heart attack management.
- 7** Appropriate technology that is timely and available 24 hours, with prompt backup interpretation. These include enzymes (CPK/MB, myoglobin, and Troponin), ST monitoring, technetium sestamibi, nuclear testing, echo, and stress testing.

EHAC's goal is to have a CPC in every U.S. hospital (more than 700 so far), to provide the penetration and hospital connection necessary to reach patients with acute myocardial infarction and acute myocardial ischemia early enough to intervene and introduce prevention practices. The strategy is to prevent disease and promote health.

If you are interested in the evolution and changing dynamics of Chest Pain Centers with emphasis on cost effectiveness and critical pathways contact:

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<http://www.chestpain.org>
<http://www.chestpaincenters.org>
<http://www.cped.org>

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A multidisciplinary approach to chest pain evaluation and management

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ABSTRACT: *Chest pain evaluation centers in emergency departments efficiently exclude acute myocardial infarctions but are limited in identifying patients with acute cardiac ischemia (ACI) without infarction. Short-term prognosis is similar for patients discharged with ACI and for those who had an infarction. Subsequently, for suspected ACI, cardiology or primary care is consulted, providing follow-up coronary angiography or stress testing, but often requiring repeat labwork and resulting in significant time delays. A coordinated approach to chest pain management—an early multidisciplinary effort—provides an efficient and cost-effective approach to chest pain evaluation and management, while reducing potentially dangerous delays in identifying ACI.*

Each year, 5,500,000 people present to emergency departments (EDs) with chest pain. The estimated cost of diagnosing this patient population approaches \$10 billion annually, demonstrating the need for efficient chest pain management.¹ Successful chest pain evaluation centers (CPECs) share several points in common: streamlined pathways, dedicated personnel, improved patient treatment strategies, and efficient time management, all of which translate to improved care, reduced medicolegal risk, and ultimately, lower costs.

“Ruled in” myocardial infarction (MI) from the ED signifies a total loss, with myocardial salvage the only remaining objective. The time to detect and manage chest pain is *before* an MI occurs. Heightened awareness of the “missed MI” provides the stimulus for the practicing physician to answer the question, “Was there a myocardial infarction?” This question can be answered by an ED CPEC less expensively, and certainly more efficiently, than by any other specialty.²

Much time and effort have been devoted to developing effective risk stratification algorithms for acute myocardial infarction (AMI). These pathways include extensive cardiac enzyme panels, treatment protocols for essential medications (e.g., aspirin, oxygen), and early intervention when MI is first identified.

Failure to identify acute cardiac ischemia (ACI) presenting to the ED may be devastating. The 6- to 24-month prognosis for ischemic chest pain not resulting in infarction is similar to that of infarction.³ An estimated 5% of patients with MIs (more than 35,000 annually) are mistakenly discharged from hospitals. On average, 16% of these patients subsequently die. Furthermore, missed MIs result in 21% of ED malpractice awards.² Since as few as 2% of ED complaints are initially identified as AMI, a second evaluation step may capture more atypical presentations for AMI; identify patients with ACI; and further reduce morbidity, medicolegal risk, and overall cost.

A second equally important question regarding chest pain evaluation must be asked when a patient presents with chest pain without an MI: "If it was *not* a myocardial infarction, then what was it?" Here, a multidisciplinary approach to chest pain is most efficacious. Follow-through stress testing from the ED appears to be cost effective, with one study demonstrating a cost savings of 62% per patient.⁴ Fifty-five percent of those who completed the ED "rule-out MI" in this trial were later identified to have ischemic heart disease by follow-up stress testing. Commitment from hospital administration, emergency medicine, cardiology, primary care physicians, and all ancillary personnel—a true multidisciplinary approach—best accomplishes the task of detecting heart disease.

Barish et al. present a "re-engineered" CPEC design considering representatives from all departments for a chest pain screening center.¹ The "process owners," those with a stake in the center, must include the executive branches of hospital administration, including the executive vice president, director of operations, financial officer, the chairs of the governing medical bodies (director of emergency medicine, director of medicine, chief of cardiology), physical plant (director of clinical laboratories, facilities management), and public relations (director of marketing, quality assurance, budgeting). The management team must include representation from clinical laboratories, cardiology, emergency medicine, and nursing.

Perhaps the biggest challenge in a multidisciplinary approach to chest pain management is assembling the key players. A team approach emphasizes a mission focused on two "customers": patients and physicians.¹ Patients are clearly the primary customers. Their comfort is important, achieved with quiet observation areas, a consistent approach to patient care, well-informed nurses, and a focused ancillary staff. One trial notes that patients prefer CPEC observational units and their rapid diagnostic capabilities to traditional inpatient admissions.⁵

The secondary customers are the physicians who follow the patient through the chest pain protocol in the ED, working with the emergency medicine staff to arrange follow-up stress testing and managing the patient as an outpatient. Today's CPEC must consider the primary care physician an extension of the cardiac pathway. Valuable information about the patient's medical history and prior cardiac workups, as well as results and disposition from the present chest pain pathway, must be communicated between the ED and the primary care physician.

Cardiology must also be integrated into all CPEC pathways. Input from this specialty is essential for developing diagnostic strategies for difficult cases. The choice of cardiac catheterization with angioplasty versus administration of thrombolytics to patients with myocardial infarction must involve cardiology. Yet, what of the patient with nonspecific ST-T wave abnormalities on electrocardiogram (ECG) and a significant cardiac history, but a nondiagnostic cardiac enzyme panel? In this scenario, although appropriate studies are essential to characterize chest pain, "no single test will supplant judgment in making the appropriate [treatment] decision."⁶ There are no adequate studies to support a singular approach to atypical chest pain. With a multidisciplinary CPEC team, the cardiologist would have been involved early in this patient's management. Chest pain management in the ED must be "multidisciplinary and based on evidence for diagnostic accuracy, clinical impact, and cost effectiveness."⁶ This approach also offers an opportunity for early intervention and risk stratification.

Clinical laboratories, registration, ECG technicians, house-keeping, and others must also be recognized as critical members of the CPEC team. Quality assurance monitoring from door to drug is well documented for AMI. Delays in obtaining cardiograms, processing laboratory tests, assigning beds, or registering patients slow diagnosis and management. Representation of ancillary services on the CPEC team is essential for timely and efficient patient care.

Myocardial infarction represents a loss to the patient, community, and society, all of whom share the staggering costs of long-term rehabilitation, lost work, and income. The efficiency of traditional ED CPECs rests on answering a single limited question: Was there an MI? A multidisciplinary approach answers the more expansive question about the patient's cardiac condition. Chest pain follow-through diagnosis and management from the ED appears costly and unnecessary. However, the long-term benefits of avoiding MI in a patient with ACI are obvious. A multidisciplinary approach provides a seamless transition from "rule-out MI" management to "rule-out ACI," eliminating redundant chest pain workups and giving the best service to the patient and health care providers.

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Current state of the art in the management of patients with acute myocardial infarction and ischemia within the Maryland Emergency Medical Service system

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ABSTRACT: *Maryland has a pioneering and sophisticated system of prehospital emergency medical care. Approximately 44,000 patients with chest pain and/or acute myocardial infarction were transported last year by emergency medical service (EMS) providers who also responded to approximately 5,000 out-of-hospital cardiac arrests. Funding has recently been prioritized to enable EMS providers to be trained and equipped with automated external defibrillators. Future consideration may be given to purchasing defibrillators with integrated 12-lead electrocardiographic capability. These devices have been shown to reduce the time required to initiate thrombolytic therapy once in the emergency department. There are presently no data in the United States that support the prehospital administration of thrombolytics.*

Maryland's Emergency Medical Service (EMS) is a pioneering system, one that should inspire pride. Even before most knew what EMS was, events were happening in Maryland. For example, cardiopulmonary resuscitation (CPR) was invented in Baltimore. Dr. William Kouwenhoven was at Johns Hopkins developing the defibrillator. He had dogs in cardiac arrest that were instrumented. While measuring the paddle pressure on their chests, he noticed the arterial pressures of the dogs spiked as he applied force. He took that a little further and eventually developed the CPR model.¹ Dr. Peter Safar, also at Hopkins at the time, said that raising the arms up over the chest to produce artificial respirations was not effective. Consequently, he recommended going back to mouth-to-mouth techniques that, in fact, were practiced in the 18th century.² Not long thereafter, the National Academy of Science combined Dr. Kouwenhoven's discovery of chest compressions with Dr. Safar's mouth-to-mouth techniques and developed CPR.

The first prehospital save from sudden cardiac arrest documented in medical literature was in Baltimore in 1960.¹ Johns Hopkins University had an early model defibrillator in the emergency department (ED). Not being

very portable, it was not going to travel to the patient. However, Hopkins had just taught the fire department CPR, so when the firefighters came upon a patient with cardiac arrest, they performed this life-saving technique. They then took him to the hospital, plugged in the defibrillator, and shocked him. He lived for many more years.

About five years later, when the device became portable enough to use in the field, Dr. Pantridge demonstrated its effectiveness in ambulances in Belfast, Ireland.³ Shortly thereafter, in 1969, the United States started to develop paramedic programs and EMS evolved from there.

Besides sudden cardiac death, there was another issue driving the development of paramedic programs in the United States, and that began in Maryland as well. A 1966 white paper by the National Academy of Science entitled *Accidental Death and Disability: The Neglected Disease of Modern Society* pointed out a number of problems with respect to trauma care in America. Dr. R. Adams Cowley at the University of Maryland was instrumental in advancing the concepts of prehospital trauma care and trauma systems in the state. Thus, Maryland developed a wonderful trauma system on his premise of the Golden Hour, which is that people who are severely injured basically have a finite amount of time to get to a trauma center.

Ultimately, it was the fire service in Maryland that picked up the responsibility for prehospital care. Maryland's rich legacy is that of an active, involved fire service throughout the state. About 75% of fire service providers in Maryland are volunteers. Approximately 25% are career providers, but that 25% handles nearly 50% of the call volume. Baltimore City is the only all-career jurisdiction; the rest are either a hybrid of career and volunteer or predominantly volunteer.

In Maryland, there are two levels of basic and two levels of advanced life-support providers. The basic life-support (BLS) providers are first responder and basic emergency medical technician (EMT). Maryland is currently transitioning from the original EMT, which was called the EMT-A, to the new EMT basic, called the EMT-B. Basic EMTs splint, provide oxygen, control bleeding, do emergency childbirth, and, until recently, in terms of the management of cardiac arrest, could only do CPR. In the EMT-B education, automated defibrillation has been added as part of the core curriculum, and along with the traditional EMT-A skills, additional proficiencies augment the EMT-B training. They can now assist patients with certain medications, such as nitroglycerin and albuterol inhalers, and they can give oral glucose, charcoal, and ipecac.

The two levels of advanced life-support (ALS) providers are the cardiac rescue technician (CRT) and the paramedic, the latter being the highest level EMS provider in Maryland. These individuals can do all the things the basic EMT can do but can, among other things, defibrillate manually and give certain intravenous medications.

The hours of training required are 40 hours for a first responder, 110 hours for the EMT-A, and 125 hours for the new EMT-B. The CRT requires about 250 hours and the EMT-P nearly 500 hours. Each level approximately doubles the training requirements.

Maryland also has a wonderful statewide system of hospitals and trauma centers. Forty-nine emergency departments around the state receive the bulk of the patients that EMS transports. There are nine trauma centers in the state, including the Shock Trauma Center in Baltimore, which is the highest level of trauma center in Maryland. Johns Hopkins is the "academic-level" trauma center; the rest are area-wide trauma centers. Uniform statewide protocols exist for triaging patients to these trauma centers.

Maryland also boasts specialty referral centers, facilities designated as being able to provide special care, with triage protocols for when a patient should be taken there or transferred there from another hospital. Specialty centers include locations for pediatric trauma, burns, hand injuries, perinatal care (new to the state), neurotrauma, hyperbaric treatment, and eye trauma. There is also current consideration of several additional specialty centers: cardiac centers, where certain hospitals are designated as expert in terms of cardiac interventions, and stroke centers. With regard to the latter, the need for specialized stroke care is another issue of emerging prominence.

In Maryland, the police continue to play a significant role with respect to the statewide EMS program. The Maryland State Police have eight helicopter bases around the state. Helicopters can transport people from trauma scenes to the hospital and also do interfacility transfers. Most of what the state police helicopters do is trauma scene care with transport to a trauma center. They also provide law enforcement and rescue services.

Another important aspect of Maryland's EMS is a comprehensive statewide medical communications system, which enables providers in the field to talk to doctors at the hospital. Maryland is one of few states with a statewide system of medical communications. EMS providers can talk to doctors at the closest hospital or, through a system of statewide microwave relays, to a trauma surgeon, cardiologist, pediatrician, or whomever they need. This communications system is run from Baltimore and broken down into two areas: the EMRC and SYSCOM. The Emergency Medical Resource Center (EMRC) is the radio communications center that links the ambulances to the physicians in the hospitals in central Maryland. System communications (SYSCOM) is the dispatch and operations center for the helicopters. Everybody works together in a single room. There is also a network of dispatch centers around the state in each jurisdiction, and comprehensive work is being rendered on a two-year implementation program so that every dispatcher in the state of

Maryland is trained as an emergency medical dispatcher (EMD). EMDs can perform medical interrogation and give prearrival instructions, including instructions to a caller on how to perform CPR prior to the arrival of a public safety unit.

Taken together—including the public-safety providers, communications network, emergency departments, trauma and specialty centers, and hospitals—the Maryland system is a very complex system that saves lives.

Maryland Institute for Emergency Medical Services System's (MIEMSS) role is to support the EMS Board and State EMS Advisory Committee that was established a couple of years ago. The board sets the regulations, and State Emergency Medical Services Advisory Council is the principal advisory body to the Board and MIEMSS. For years, MIEMSS has been certifying the basic EMTs. The Board of Physician Quality Assurance certifies the advanced life support providers. Next year, MIEMSS will consolidate those functions and will either certify or license all the EMS providers through the EMS board.

MIEMSS is working hard on quality management at a state level, while trying to encourage it at the regional and local levels as well. MIEMSS believes that quality management is a big part of the future of EMS. MIEMSS provides grants for equipment and education. We purchase and distribute a number of automated external defibrillators and defibrillator/monitors each year, although they are still in the implementation phase of getting an adequate number of defibrillators in the hands of EMS responders. One of the questions for MIEMSS now is at what point can we slack off on purchasing defibrillators and look at the purchasing of 12-lead electrocardiogram-capable defibrillators for the field. MIEMSS' responsibilities also include public information and education.

In terms of call volume, in fiscal year 1996, Maryland experienced approximately 500,000 EMS responses, 270,000 of which resulted in transports. There were 251 neonatal transports and a little over 4,000 medivacs. The bulk of the medivac flights were from scene to trauma center. Statewide, EMS has an average response time of 8.6 minutes. Response time is defined as the period from the moment the 911 call comes in to EMS' arrival on the scene. The bulk of EMS patients are what is called Priority 3, which basically means stable. Priority 1 is the most severely ill or injured patient; Priority 2 indicates the patient is potentially unstable. In fiscal year 1996, Priority 3 patients amounted to 52% of transports; about a third of transports were Priority 2 and close to 15% were Priority 1.

What are the procedures most commonly done by EMS? Oxygen. Almost half the patients who are transported get oxygen. Spinal immobilization follows as the next most frequent procedure, and then controlling bleeding, splinting, suctioning, and obstetric deliveries. What kind of medications are given? Interestingly, the most frequently

administered medication is nitroglycerin. Thirteen thousand and seventy-five doses of nitroglycerin were given out in fiscal year 1996. Chest pain is one of the most frequent transports.

The protocol for acute myocardial infarction (AMI) or chest pain is fairly straightforward. Basically, it consists of oxygen, a cardiac monitor, and an intravenous line. Providers carry lactated ringers which is usually administered slowly. The providers can give nitroglycerin sublingually. Starting in July, both advanced life support and basic life support providers can give a dose of nitroglycerin, without having to start an intravenous line initially, if the patient has a preexisting prescription, has taken nitroglycerin before, and is otherwise stable. Atropine and lidocaine are given as needed if the patient is symptomatic. If the patient is asymptomatic and the provider feels that atropine or lidocaine is indicated, they can call in for medical direction and get an order. To give morphine sulfate, medical consultation is required. The policy now is to transport the patient to the closest emergency department.

As previously mentioned, one of today's burgeoning issues is the 12-lead electrocardiograms (ECG) in the field. This is a new kind of technology, integrating 12-lead ECG machines into the standard monitor/defibrillator. It costs about \$4,000 or \$5,000 more than a standard monitor/defibrillator. There are still some issues in terms of their size and portability, but these devices are now basically monitors, defibrillators, 12-lead ECGs, external pacers, and there is a growing consensus to include pulse oximeters and end-tidal CO₂ detectors. Essentially, this is a portable intensive care unit. In terms of the 12-lead ECG, it only takes about five to ten hours of training to enable a paramedic to perform an ECG in the field. At this time, four jurisdictions — Prince Georges, Montgomery, Howard, and Baltimore Counties — are doing them.

The Acute Cardiac Ischemia-Time Insensitive Predictive Instrument (ACI-TIPI) is also being considered. A project of Dr. Harry Selker from Boston,⁴ with the ACI-TIPI, you have a 12-lead ECG to which you add age and other factors to determine the percentage likelihood that the patient is having an ischemic event.

Clearly, using 12-lead ECGs in the field is a feasible and easy undertaking. Successful ECGs can be performed on nearly 95% of the patients encountered with chest pain. The criteria for a patient getting a 12-lead ECG in the field are that they be an adult with chest pain, normotensive, and with no malignant dysrhythmias. Does doing a 12-lead ECG in the field add to scene time? The answer is yes, but not much. On the average, probably three to five minutes are added. Putting a 12-lead ECG program in place involves a fair amount of quality assurance monitoring to make sure that scene times are not exceeded and other issues are addressed. One of the benefits of 12-lead

ECGs in the field is that it assists the base station physician. A standard 3-lead ECG strip transmitted over the radio, with a history provided by the prehospital provider, gives a specificity of 68% with respect to diagnosing an AMI. If the provider sends a 12-lead ECG with a history, that increases to 95% specificity. To date, there are no proven clinical outcome benefits of 12-lead ECGs in the field, although 12-lead ECGs reduce the door-to-drug time for thrombolytics in the ED for patients with AMI. In the Cincinnati Heart Study Project, if an AMI patient came in by private vehicle, on the average, it took 64 minutes to get thrombolytics on board. If they came in by ambulance, the time was 55 minutes. Via an ambulance that did a 12-lead ECG, but had not transmitted it to the ED, the time was 50 minutes. If the providers did a 12-lead ECG and transmitted it ahead to the physician in the ED over a cellular telephone, then it reduced the door-to-drug time to 30 minutes. While there is a clear drop in the door-to-drug time, it has not been statistically proven that the time saved provided any clinical benefits.⁵

The Myocardial Infarction Triage and Intervention (MITI) study in Seattle in 1989⁶ was a study of prehospital thrombolytics. The investigators noted that in the control group of AMI patients identified but not treated in the field, the ED door-to-drug time was reduced from 60 minutes to 20 minutes. So, the overall time benefit of administering thrombolytics in the field was reduced because the control group had a significant reduction in the ED door-to-drug time.

In summary, 12-lead ECG can be done in the field. They clearly reduce the door-to-drug time in the ED, somewhere between 20 to 40 minutes on the average. Because this time difference is a relatively modest decrease, it has not yet been proven that it reduces mortality or improves outcome. It is also important to note that reducing the time to thrombolytic therapy for AMI patients with 12 lead-ECGs in the field requires a community effort. The EMS system has to be geared up and the hospital has to be working with them.

As previously discussed, one of the things that really spurred the development of EMS in this country was the syndrome of sudden cardiac death. In Maryland, EMS responds to about 5,000 cardiac arrests a year. In fiscal year 1996, 41% of the cardiac arrests EMS responded to were witnessed arrests, and almost a quarter of them had CPR on EMS arrival. Data from the prehospital patient care report database, shows about 40,000 records in which patients either had chest pain or were thought to be having an AMI by the EMS provider. Adding 10% for one jurisdiction for which data are not available, EMS takes care of and transports an estimated 44,000 AMIs or chest pains a year. One of the questions currently being decided is whether to continue to invest heavily in getting

AEDs and more defibrillators in the field or whether to start buying 12-lead ECGs that cost much more. Having to buy 12-lead ECGs at this time would effectively reduce the number of defibrillators that are continuing to be distributed statewide.

Recent Health Services Cost Review Commission (HSCRC) discharge data from Maryland hospitals indicate, using the ICD-9 categories for AMI of 410.XX1 (where 1 stands for the initial episode), there were somewhat fewer than 15,000 discharges. That included about 5,500 SEMIs. Not including the SEMIs, that leaves around 9,000 AMIs. In most EMS systems, about 50% of patients with AMIs get to the hospital by EMS and about 50% get there by private vehicle. So, one way of estimating how many patients EMS in Maryland transports per year that are potential candidates for either thrombolytic care in the field or thrombolytic therapy when they get to the ED is to take 50% of the AMIs, or roughly 4,500 patients. That is a high number, since there will be contraindications in a number of the potential candidates.

There is one other way of calculating that number. In the MITI study, it was found that only about 5% of the patients who are transported by EMS with chest pain are ultimately determined to be candidates for thrombolytic therapy. Calculated that way, 5% of 44,000 is about 2,200 candidates for thrombolytic therapy. Consequently, somewhere between 2,200 and 4,500 patients transported by EMS in Maryland last year were AMI patients who were candidates for thrombolytic therapy. Opting to use the higher number, 4,500 patients transported by EMS in Maryland last year were candidates for thrombolytic therapy.

European studies have demonstrated that if the pain-to-drug time is reduced for thrombolytic therapy in AMI patients by one hour, one can expect a 1.5% increase in the three-month survival rate.⁷ On the average, U.S. studies have demonstrated a time savings of about 34 minutes with prehospital 12-lead ECGs. If one estimates high on both the number of AMIs who are potential candidates for thrombolytic therapy and the survival rate, 68 lives (1.5% of 4500 patients) could theoretically be saved per year if the pain-to-drug time was reduced by one hour with 12-lead ECGs in the field.

Nearly 5000 cardiac arrests occur each year in Maryland. In well-developed EMS systems, they can achieve about a 25% to 30% survival rate if the system is optimally configured. So the goal, and the potential, in Maryland is to save about 1250 patients from sudden cardiac death. To do that, AEDs must be placed in the hands of all basic responders and monitor/defibrillators must find their way into the hands of all paramedics. Thus, for the moment, the priority should be that of ensuring a timely and appropriate response to sudden cardiac arrests, since that is where the potential to save the most lives (68 vs 1,250 lives per year) exists. Once a reasonably comfortable distribution of

AEDs and defibrillators is reached in the public safety community, funding should be considered for the purchase of 12-lead ECGs for field use.

Another prehospital cardiac care question that has been discussed for a few years is whether to start administering thrombolytics prehospital. In the MITI study, which is one of the few studies of prehospital thrombolytics completed in the United States, by doing thrombolytics in the field (which involved doing a 12-lead ECG, talking to the ED, going through a list of questions looking for contraindications, etc), providers could save 33 minutes in pain-to-drug time. In other words, they could get thrombolytics on board 33 minutes earlier than if they transported the patient and did it in the ED. That 33 minutes was not enough for them to prove any benefit in terms of mortality or morbidity.

There are some interesting studies outside the United States. In Scotland, the Grampion Regional Study⁸ got a two-hour benefit by giving thrombolytics in the field. Yet, in sharp contrast to the United States, the average hospital time door-to-drug was 240 minutes. Consequently, the Grampion study really does not have much application in the United States. There is also the European Myocardial Infarction Project (EMIP)⁷ that had over 5000 patients randomized for a study of prehospital thrombolytics. In this study, there were physicians on board the ambulances, and the 30-day overall mortality for AMI patients was reduced, but not statistically, from 11% to about 9.7% if the thrombolytics were given in the field. The cardiac mortality was reduced from 9.8% to 8.3%, and that was statistically significant. This is the 1.5% maximal benefit used to calculate the potential impact of 12-lead ECGs in Maryland. One of the issues clearly discussed in detail in the EMIP study was that when thrombolytics were given in the field, they saw the same problems in the field that are seen when given in the ED. Dysrhythmias were common, the patients became hypotensive, they became bradycardiac. Those situations can be difficult to manage in the field, particularly when one is alone in the back of that ambulance. Even when there are two people, it is not like being in an ED. In most EDs today, there are stretchers that allow staff to gather around, and there are teams to bring in to help deal with these complications as they happen.

One of the things generally known from the studies that have been done on thrombolytics is that there must be about an hour of reduction in pain-to-drug time to show a statistical benefit in outcome. With respect to prehospital thrombolytics, in the United States under the best of circumstances, that kind of time benefit is not likely to be gained in an urban EMS system with relatively short transport times and EDs that are generally geared up to rapidly evaluate and treat patients with AMI. To save an

hour of time in the United States, one would have to look at rural systems with longer transport distances, and at the present time, most rural systems lack the structure and sophistication to develop prehospital thrombolytic programs. That is one of the reasons that these European studies of prehospital thrombolysis are not generally applicable to the United States. Another important issue is that of making a diagnosis of AMI in the field without a physician present. While it has been done in several U.S. studies, it is not at present accepted as appropriate except in a few fairly sophisticated EMS systems with intensive medical oversight. Although additional U.S. data on prehospital thrombolysis may offer further insight, the MITI study and the smaller study in Cincinnati did not demonstrate any benefit, and there just has not been much interest.

One final issue is the triage of patients with chest pain. Presently, they are transported to the closest ED. There has been some dialogue about whether patients should, in fact, be going directly to a designated chest pain or cardiac center, generally a place that has interventional cardiology. The position now is that since there are no clear data on the benefit of this, it makes sense to continue to transport to the closest ED. However, there is interest in continuing to look at the issue, searching for more data. At this point, it is unknown if a patient is better off getting thrombolytics in the closest ED and then transferred, if necessary, to a center with more advanced cardiovascular services, or whether, despite a longer travel time, the outcome would be better if the patient were transported directly to the cardiovascular center.

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An eight-month evaluation of prehospital 12-lead electrocardiogram monitoring in Baltimore County

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ABSTRACT: *The purpose of this evaluation was to determine if a prehospital 12-lead electrocardiogram (ECG) led to a delay in transportation when compared to non-12-lead ECG, and if it led to improved "door to drug" time. A quasi-experimental design was used to compare on-scene times for suspected acute myocardial infarction (AMI) patients; a comparison was made between advanced life support (ALS) units without 12-lead ECG and those who were augmented by equipped EMS district officer units. Our control group had an on-scene time of 22.05 minutes; the mean on-scene time for the experimental group was 21.85 minutes. The results of this study indicated that 12-lead ECG acquisition by district officers did not extend the on-scene time.*

The Baltimore County Fire Department Emergency Medical Services (EMS) division provides emergency medical service to over 700,000 residences. Baltimore County is 610 square miles of farmland, heavy industry, light industry, and governmental offices. The delivery of EMS is provided by a combination of career and volunteer providers who staff 42 transporting medic units, eight district officer vehicles, and ten paramedic engine companies. In 1996, the EMS division responded to over 70,000 incidents. Of these incidents, cardiac-related illnesses accounted for approximately 23% of the emergencies. To address these cardiac emergencies, the effectiveness of a 12-lead electrocardiogram (ECG) program in the area needed to be determined.

The 12-lead ECG program was implemented in early 1996 through the collaborative efforts of Saint Joseph's Hospital and the Baltimore County EMS staff. Saint Joseph's Hospital donated five Marquette Responder 1500 monitors. Before placing the monitors in service, a focus group developed policy, procedures, and quality assurance measures. The

focus group also sought approval from the jurisdictional, regional, and state medical directors.

Policy

At present, the 12-lead ECG program is limited to persons certified at the National Registry emergency medical technician paramedic (EMT-P) level. Before utilizing the device, each provider must attend a nine-hour training course that provides information on basic myocardial infarction detection/recognition and equipment familiarization. In addition, a four-hour continuing education program has been developed, covering topics such as anatomy and physiology, procedures related to chest pain management, and current changes in chest pain management. An ECG case management review program, consisting of various 12-lead ECG rhythm strips for interpretation, was also developed by the fire rescue academy.

Candidates for 12-lead ECGs include patients meeting the Maryland state medical protocol for AMI. Other cases for which a 12-lead ECG may be beneficial include arrhythmias, overdoses, and strokes, as well as cases categorized as such by the field providers, and those consulting physicians feel may benefit from its use.

Procedure

The paramedic should obtain the 12-lead ECG at the patient location. The EMS providers must consider environmental hazards that may impact patient or provider safety. The EMS providers perform patient assessments and treat life-threatening emergencies. After the Marquette 1500 responder acquires the ECG, the providers print a hard copy. As patients are prepared for transportation to the medic unit, the district officer may transmit the ECG via cellular or land phone to a receiving facility. While en route to the hospital, the paramedics should complete a thrombolytic survey form. Upon arrival at the emergency department (ED), the paramedics should present the out-of-hospital 12-lead ECG and the thrombolytic survey form to the ED physicians.

Quality assurance measures

The quality assurance committee is responsible for evaluating the training programs and course content for accuracy and quality. They are also responsible for developing a database, opening communications with local hospitals, developing a thrombolytic survey form, and seeking approval from the ED physicians and medical directors.

Data collection and results

Data for this design was collected between July 15,

1996, and March 1, 1997. The purpose of our study was to determine the outcome of the following: 1) was the on-scene time delayed, and if so, by how much and for what reason? 2) how accurate were the computer-generated 12-lead ECG and interpretations? and 3) did we reduce the "door to drug" or catheterization time?

Was the on-scene time delayed, and if so, how much and for what reason?

On-scene time was the most important question to be answered. Other studies have shown an increased on-scene time of three to five minutes.

Method and procedure. A quasi-experimental design was used to compare on-scene times for suspected acute myocardial infarction (AMI) patients in two situations: advanced life support (ALS) units without 12-lead ECG, and those who were augmented by equipped EMS district officer units. For this study we had a control group that included 117 patients with a chief complaint of chest pain. They all received standard cardiac care without 12-lead ECG. The experimental group included 70 patients, each receiving standard cardiac care plus a 12-lead ECG. The data were collected from a convenient sample of EMS reports generated over an eight-month period. Data were analyzed using GB Stat 6.0 statistical software. Statistical evaluation included descriptive statistics and a Student's 2-tailed *t* test.

Results. The results demonstrated a mean on-scene time for the control group of 22.05 minutes, with a standard deviation (SD) of 8.06 and a standard error (SE) of .745. Mean on-scene time for the experimental group was 21.85 minutes, with a SD of 7.37 and a SE of .907. The Student's 2-tailed *t* test revealed a 95% confidence interval of -2.12 to -2.52. The critical *t* -value was .173 with a probability of .863. These results revealed a statistically significant study.

Conclusion. In our study, the results indicated that 12-lead ECG acquisition by our district officers did not extend on-scene time.

How accurate were the computer-generated 12-lead ECGs and interpretations?

Another concern was the reliability of the computerized 12-lead ECG diagnoses when compared to final diagnoses and patients' discharge summaries.

Method and procedure. Saint Joseph's Hospital received 32 patients during this study. Data were compared to the "patient outcome data" provided by Saint Joseph's Hospital.

Results. The results for out-of-hospital 12-lead ECGs suggested that 12 patients had a positive indication of

AMI. The hospital data confirmed that seven of the 12 patients had an AMI. The five remaining patients whose results suggested an out-of-hospital AMI were diagnosed as follows: two had congestive heart failure and left bundle branch; one arrested and did not survive; one had nonspecific sinus tachycardia changes; and one had stable ventricular tachycardia. The data also demonstrated that of the remaining 20 patients, none were misdiagnosed by the provider or the computerized report.

Conclusion. This study showed that out-of-hospital 12-lead ECGs had a sensitivity of 58% and a specificity of 100%.

Did we reduce the "door to drug" or catheterization time?

The final issue identified as a critical factor was the treatment rendered by the medical staff.

Method and procedure. With the assistance of the Saint Joseph's Hospital staff, a database was developed to identify the patients' diagnoses, types of treatment, and time of treatment. An event summary was conducted by identifying patients diagnosed with AMI and assessing their treatment modalities.

Results. Seven of the 32 patients were diagnosed with AMI. The patient outcome summary provided by Saint Joseph's Hospital was reviewed and the following results were obtained: one patient received tissue plasminogen

activator (t-PA) in less than nine minutes; one patient was transported to the catheterization laboratory within ten minutes of arrival to the ED; and three patients went directly from the ED to the catheterization laboratory. Specific time interval data was not obtained. Outcome data was unavailable for one patient.

Conclusion. The aggressive treatment provided to these patients probably had an impact on their morbidity and mortality. However, further trials will be needed to clearly demonstrate a reduction in morbidity and mortality.

Discussion

Several issues must be addressed. A substantial cost is associated with the 12-lead ECG program — each monitor costs approximately \$14,000. In addition, there is the cost of cellular phones and air usage. Training issues encompass both initial training and continued education hours. This cost could be rather substantial for a large department.

To have a successful program, it is necessary to have the support of the administration, the prehospital care providers, and the hospital and ED staff, as well as a good quality assurance program. As stated in the January 1997 *Annals of Emergency Medicine*, "studies to date demonstrate that prehospital 12-lead ECG technology is feasible and clinically practical and probably could be implemented in most established urban paramedic systems. This time savings is perceived as beneficial but has not, by itself, demonstrated a reduction in mortality." ■

NOTICE • NOTICE • NOTICE • NOTICE • NOTICE • NOTICE

NEXT CONGRESS OF CHEST PAIN CENTERS CONFERENCE

SEPTEMBER 1998

Prepare now to attend the Third National Congress of Chest Pain Centers. The conference will be held in late September 1998 in Royal Oak, Michigan.

The Congress will feature the "best practice to date" and our ability to shift the paradigm of heart attack care to earlier presentations with coronary syndromes, unstable angina, and non Q-Wave myocardial infarction patients. The highlight of the program will be presentations on the use of intravenous and oral platelet glycoprotein II-B/III-A antagonists in acute coronary syndromes.

The proceedings of this conference will be distributed to cardiologists, emergency care physicians, and critical care nurses and will be available on our website (<http://www.chestpaincenters.org>). Please e-mail us (info@ehac.org) for further information as the conference develops.

Emergency Medical Service providers' role in the early heart attack care program: prevention and stratification strategies

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ABSTRACT: *Emergency Medical Services-Early Heart Attack Care (EMS-EHAC) is a community-based program where paramedics increase the consumer's awareness about early chest pain symptom recognition. EMS-EHAC prevention, along with seamless chest pain care (between the paramedic and chest pain emergency department) can be the basis for an outcome-based study to examine the impact of advanced life support EMS.*

Studies that show the impact of care given by paramedics on the outcome of patient care must be designed to demonstrate the value and the cost benefit of providing advanced life support (ALS). Third party payers are going to examine if there are significant quality differences between ALS and basic life support (BLS) services. If significant benefits of ALS care cannot be demonstrated, the cost differences could potentially place the future of advanced life support paramedic programs in jeopardy.

A positive outcome resulting in a lower acute cardiac event, and the realization of the cost benefits from the EMS-EHAC program could be utilized by EMS management to justify or expand advanced life support programs.

Introduction

Are paramedics an endangered species? This question was recently posed by William Brown, the executive director of the National Registry of Emergency Medical Technicians (EMTs).¹ Recent changes in medicine have affected every corner of health care in the United States. With managed care's impact and changes in governmental funding, emergency medical services (EMS) will not escape financial scrutiny. The payers will

ask questions regarding the number of paramedics per unit, the additional skills that can be taught to basic life support (BLS) providers, and the claim nursing has to out-of-hospital critical care.

The most significant question yet to be answered is whether paramedics (EMT-P) affect discharged patient outcomes. The major payers have initiated reasonable and necessary technology assessment guidelines to evaluate the clinical and economic benefits of health services. The salary, training, and equipment cost differences between advanced life support (ALS) EMT-Ps and BLS EMTs are significant. Consequently, the health care payers will ask if there is a significant difference in patient outcome between the two provider groups. Shuster et al., from McMaster University in Hamilton, Ontario, studied this issue for the Hamilton-Wentworth region of Ontario, Canada. Over 3,000 patients with acute cardiac illness transported by either ALS or BLS crews trained in defibrillation were studied over 3.5 years. Seven hundred and eighty three patients sustained myocardial infarctions (MI), and there was no difference in the proportion of people discharged alive between the two study groups. There were also no significant differences in the length of hospital stay. The authors' conclusion was "in an urban setting with short (< 10 minutes) average transport times, the availability of prehospital paramedic care does not affect the occurrence of MI, length of hospital stay, or mortality of patients presenting to the EMS system with cardiac illness." Shuster was disappointed at not being able to demonstrate the benefits of paramedic care since prehospital ALS attention has historically been assumed to provide superior results.^{2,5} He also states that the outcome measures may not have been totally appropriate. This type of study could significantly hinder the progression of ALS paramedic care, since it demonstrates an equally effective method of care at a lower cost.

EMS researchers must design and implement outcome-based studies with significant clinical results that can justify the additional costs. The current work practices of EMS systems should be investigated and adjusted for maximal efficiency. This two-part article introduces a new concept called Emergency Medical Services - Early Heart Attack Care (EMS-EHAC).⁶ This is an extension of the early heart attack care program developed by the St. Agnes HealthCare System modified to have an EMS focus. Implementation of EMS-EHAC, along with a comprehensive acute chest pain stratification and treatment program, has the potential to significantly impact the rate of myocardial infarction by imparting a message of early symptom recognition and an urgency to act during periods of acute symptoms. Prospective community epidemiological and hospital discharge outcome studies could be designed to measure the impact of this prevention/strati-

fication/treatment program, along with health-economic analysis of the change. Effective studies that demonstrate positive health care outcome and significant health care savings confirm the value of ALS and are the necessary tools to ensure the future of paramedics and EMS.

SECTION I

Prevention strategies: using prevention programs as a tool to increase the value of EMS

Prevention

Prevention programs have been designed, launched, and evaluated for efficacy many times in recent history. Programs for areas such as reduction of cavities, forest fire prevention, human immunodeficiency virus (HIV) transmission, and drunk driving have, in each case, been quite effective in reducing their respective prevalence. Public health education projects in these areas have been well funded through governmental or private agencies, and media campaigns have promoted the messages so thoroughly that essentially every citizen has had repeated exposure to the messages. Having continuous exposure to the prevention messages should not only facilitate behavioral changes, but also maintain the positive results.

Marketing prevention programs

A product can make an impact on preventative care when advertising campaigns spread the prevention message while branding their particular product. For example, Trident® chewing gum's "four out of five dentists surveyed recommend sugarless gum for their patients who chew gum" campaign. Similar messages are delivered through Crest or Colgate toothpaste advertisements. These campaigns were not only successful for the manufacturers, but they also, working in concert with reminders from the dental community for prevention visits, contributed to a significant decrease in the prevalence of tooth decay over the years.

One of the most successful publicly funded advertising campaigns is Smokey The Bear's "only you can prevent forest fires" message. Fire departments have had prevention duties for many years. These functions have helped develop building codes, create public education programs, and minimize fires through the installation of devices like sprinkler systems. With the recent invention of residential smoke detectors, a strong public/private advertising program has formed; most homes are now equipped with smoke detectors. Although smoke detectors have been credited with saving many lives, continual messages must be sent to the public to have the detectors checked for functionality, since lives are frequently lost in dwelling fires where investigators determine that the house had either no detector or a detector with dead batteries.

Today's fire department

In this country, 911 calls for EMS are delivered by several types of provider agencies. The responding units could be privately owned, hospital based, volunteer, or municipally operated. Large, consolidated private ambulance companies have actively been seeking and winning municipal EMS contracts. The economics of these contracts relieves the citizens of the tax burden and liability of providing EMS, while the private contractor is able to bill for patient transportation.

Many fire departments have been noticing the changes in the mix of emergency responses. Successful prevention efforts have resulted in a significant drop in actual fire incidents. While the firefighting load is dropping, these departments are now experiencing rapid growth in their medically related responses. In Howard County, Maryland, EMS incidents comprise greater than 60% of all emergency responses.⁷ This change in response pattern has caused the fire department to change its hiring requirements. For the first time, applicants for the recent recruit class had to be paramedics. Noticing the trend, many senior career firefighters have completed, or plan to enroll in, paramedic classes. Staffing changes include placing paramedics on fire engines to increase the department's ability to respond rapidly with ALS capability.

Most fire departments have active prevention programs that are either centrally administered or maintained at a station level. For example, the Howard County Department of Fire and Rescue Services has a prevention section that spends approximately 80% of its time on fire prevention activities (i.e., code enforcement, inspections, public education) and 20% on safety inspections (i.e., Occupational Safety and Health Administration-type examinations). The safety inspections are considered injury prevention programs, but this unit does not concentrate on other medical prevention programs.

EMS prevention in Howard County is limited to the individual EMS company's participation at health fairs, blood pressure screening programs at the local shopping center, or standing by at large gatherings (e.g., high school football games). These programs are sporadic and not associated with continual prevention programs. There currently are no outcome-based prevention studies underway.

As health care costs and pressures on municipal spending increase, the EMS field, particularly ALS, is going to be scrutinized for significant beneficial clinical outcomes and cost effectiveness. The concern being expressed by EMS leaders is that a "squeeze" is happening between enhanced basic life support EMTs (EMT-Basic trained in more advanced skills like defibrillation), the return to rapid transportation for certain high-priority conditions (e.g., MIs and strokes), and potential for nursing to compete for the critical

TABLE 1. ACC/AHA EMS guidelines for the chest pain patient

Class I: (definitely helpful)

- 911 access
- system availability of defibrillation
- triage of ischemic pain

Class IIa : (acceptable, probably helpful)

- first-responder defibrillation
- health care providers educate patients/families about signs and symptoms of acute MI, accessing EMS, and medications

Class IIb: (acceptable, possibly helpful)

- 12-lead telemetry
- prehospital thrombolysis (special circumstances)

care component of out-of-hospital medicine. The term "expanded-scope paramedic" has been in the literature recently.^{1,8} Expanded scope is a set of additional primary care skills utilized by paramedics between emergency calls. One permutation of the expanded scope concept comes from Jack Stout of MedTrans, who is developing programs where paramedics are crossed-trained as physician assistants.¹ The dually trained providers will be working out of ambulances doing nonemergent home visits, while remaining in service for emergency calls. The expanded scope paramedic is trained to administer vaccinations, staff rural health clinics, and make house calls on certain patients. The debate regarding this role for paramedics is ongoing, since this may be seen as an encroachment on the role of the home health care nurse or physician assistant.

Expanded-scope EMS certainly has the potential to increase the value of paramedics. The term "value" in the business world is viewed as the sum of product quality (or benefit) and product price. In EMS, quality has always been regarded as one of the highest virtues; however, to demonstrate value to the new payers, price becomes a term of growing significance.¹

How does one extract the value of EMS? Market conditions have caused many businesses to downsize, causing workers to perform at levels equal to or greater than before, but with fewer resources. In EMS, this has surfaced in many areas with questions like: are two paramedics needed on each ambulance? and are BLS ambulances and ALS chase vehicles necessary?

The next question may be: is ALS needed at all? Emotionally, one would agree that ALS is an important part of out-of-hospital medical care, but where are the outcome data to support its existence?

EMS is a product that has a buyer and a supplier. The buyer role is changing from public consumer to third-party payers

TABLE 2. Early heart attack care warning symptoms

Specific heart attack symptoms (prodromal angina)	Nonspecific heart attack symptoms
<ul style="list-style-type: none">• chest discomfort• chest pressure• chest ache• chest burning• chest fullness	<ul style="list-style-type: none">• weakness• sweating• nausea• dizziness

or governmental budgeting offices. Third-party payers (e.g., Medicare) use clinical benchmarking to determine if a contracted service has value over competing modalities.⁹⁻¹¹ If enhanced basic life support services can be shown to have equal clinical value through patient outcome studies, and have significant cost advantages, then BLS may be the winner.⁴ This is why paramedics must demonstrate their total value, both clinically, through patient outcome studies, and in terms of cost-effectiveness through health-economic studies. The next section introduces a prototype chest pain prevention and stratification program that, when implemented, could be utilized to help determine the value (both clinical and economic) of ALS services regarding the care of the chest pain patient. The environment is changing rapidly, and EMS must be prepared for significant paradigm shifting in order to not only survive but thrive in the future.

Chest pain, the cornerstone of EMS

Treating the chest pain patient has been the hallmark for the ALS segment of EMS. Historically, it was this type of patient who would allow the paramedics to best utilize their assessment skills and perform many of the clinical skills of the trade (intravenous drug delivery, medications, electrocardiogram [ECG] recognition, etc.). The paramedic's involvement with this patient typically starts with a 911 call for a suspected MI.

The recent ACC/AHA [American College of Cardiologists/American Heart Association] *Guidelines for the Management of Patients with Acute Myocardial Infarction* has specific recommendations for the role of EMS in the prevention, stratification, and management of chest pain patients. These recommendations are divided into classes based on the level of scientific evidence (Table 1).

The concentration of this article will be on the Class IIa recommendation of patient and family chest pain education. Most EMS programs do not include the prevention programs suggested from the AHA/ACC, even though pre-

vention has a stronger recommendation than the costly implementation of 12-lead telemetry and prehospital thrombolysis. Diagnostic and treatment recommendations are discussed later.

Prevention by intervention

The MI patient calls 911 when the signs and symptoms of a heart attack become debilitating. As this patient becomes increasingly symptomatic, the cardiac muscle is further injured. Delays in seeking medical care for this pain are not uncommon, as the patient denies that a heart attack is occurring.¹² It is not uncommon for the MI patient to have subtle prodromal symptoms before the acute event. With proper medical and general public education, these subtle symptoms could be recognized as a significant prognostication tool for an impending acute event.⁶

EMS systems and emergency departments are refining their critical pathways to deliver thrombolytic or percutaneous transluminal angiography (PTCA) therapy more quickly. The goal from the National Heart Attack Awareness Program of the National Institutes of Health estimates a life saving potential of 15,000 additional patients if the time to thrombolytic therapy is reduced to less than 30 minutes.¹³ With the rapid growth of chest pain centers, this figure is still largely unmet.¹⁴ If the recognition of prodromal symptoms can be taught with the same fervor as cardiopulmonary resuscitation (CPR) classes, the life saving potential could be as large as 150,000 patients per annum.⁶ This philosophy of early patient intervention during the pre-infarction ischemia phase is the basis of the EHAC program (Table 2). The GUSTO I study discusses the high cost of a thrombolytic save (\$200,000 per patient) versus the significantly lower cost of cardiac protection therapies.¹⁵ The life savings of early symptom awareness programs is a clinical advantage, and the potential for significant reduction in health care costs gives the EHAC program tremendous value.

EMS-EHAC (bridging EMS and EHAC)

The value of EHAC can be utilized by EMS. EMS-EHAC is a community-based program that utilizes the community-based chest pain experts (paramedics) to deliver the EHAC message to the local neighborhoods through area businesses.

The program utilizes paramedics as community chest pain symptom ambassadors. Paramedics become familiar with the concepts of the EHAC program and become trained as EHAC instruc-

TABLE 3. Potential EMS-EHAC businesses

- optical stores
- dentists
- chiropractors
- pharmacies (particularly the independent stores)
- health food stores
- barber shops
- "nail care" and beauty salons

TABLE 4. Types of EHAC monthly messages

- "Are you aware that only 40% of patients with a heart attack call 911?"
- "If you are having any of these symptoms, do not waste valuable time by calling your family physician; call 911 immediately."
- "Women are at increasingly higher risk of a heart attack. Did you know that one in four women over 65 have heart disease?"
- Future programs could address pulse and blood pressure screenings, and stroke prevention.

tors. By utilizing a method similar to community policing, the paramedics, while in service in the community, "sell" the concept to local businesses and ask for their participation in the program. Each paramedic would develop a "customer" list of approximately 10 to 15 local health-related businesses (Table 3).

The paramedic would present the program to these business owners using EMS-related EHAC tools and enlist the businesses as partners to deliver the information to their customers. The paramedic would stress the "value-added" component for the businesses, as well as the fact that their customers would appreciate the businesses' concern for their health and well-being. The health care-related businesses would then present the EHAC information in a brief, but thorough, manner to their customers. It is important to have the same paramedics visit the customers to increase the relationships through continuity.

The paramedics will visit "their customers" monthly to:

- restock the businesses with EMS-EHAC brochures
- reinforce the need for the businesses to cooperate in this program
- update the business owners with a new "message" for the month
- ask for feedback (positive and negative) from the businesses
- further develop the relationship with the businesses and the EMS organization

The monthly message is an opportunity for EMS to stress a certain local need (Table 4).

Who benefits from EMS-EHAC?

The EMS system benefits by having enhanced recognition within the community. EMS-EHAC is a marketing program that helps brand the EMS company in a similar manner to the branded toothpaste and fire prevention messages. This program also satisfies the Class IIb recommendations from the ACC/AHA regarding the education of patients and families

about signs and symptoms of acute MI. This is a cost-effective utilization of time and a minimal cost program. As Brown said, "people sitting around and getting paid for business to arrive will not survive in the future." Therefore, between calls, the paramedic can be in the community "saving lives" by delivering the EMS-EHAC message.¹

The participating businesses benefit by improving their relationship with their customer base. The businesses could advertise their participation as a competitive advantage (does your pharmacy do this?) by personalizing the EMS-EHAC brochures with the company name or creating a symptom list that could be placed on a refrigerator magnet. This also increases the name recognition of the businesses. Businesses are becoming more socially aware, and this program has the potential to significantly impact society.

The community could benefit from EMS-EHAC due to the potential for decreasing the number of MIs and deaths, the potential for less health care expenditures, reduced insurance costs, and continued earning potential. The positive clinical and economic benefits for the community increases the value of the EMS.

Evaluation of program effectiveness

Any program changes must be monitored for effectiveness by defining specific goals and an evaluation matrix before implementation. EMS studies have historically been structural and process-based. Response times to incidents and efficacy of cardiac arrest medications or airway control devices are examples of the typical prehospital study. These studies usually do not follow the patient through to discharge. Outcome studies are designed to look at the larger picture of the therapies. A positive response to a therapy upon admission to the emergency department does not necessarily translate to a measurable discharge difference. As mentioned earlier, Shuster's outcome study compared two therapeutic modalities (BLS vs ALS treatment of chest pain patients) and found insignificant differences upon discharge.⁴

TABLE 5. EHAC structure and process

Structure:

- number of paramedics working in the community
- potential number of health-related businesses
- 12-lead ECG program, rapid stratification pathways, first-responder defibrillation program, chest pain emergency department

Process:

- number of paramedics x number customers x number of presentations per month = contacts per month

Health care payers utilize these studies, along with data on costs, to influence what services are offered.

Before the implementation of the EMS-EHAC program, as mentioned, specific goals and an evaluation matrix should be developed. The process should follow the usual quality assurance pathway by first examining the structure and processes needed for the program, as well as specific, measurable goals for the outcome segment (Table 5). The program cannot proceed to the outcome segment unless there is a proper structure in place, and that process must be capable of agenda execution.

For example, Howard County has approximately 50 full-time paramedics. In this scenario, each paramedic is responsible for enlisting ten business sites. The paramedic will ask for a commitment from the business to present EMS-EHAC to its customers at least six times per business day.

- 50 paramedics x 10 businesses x 6 presentations per day = 3,000 consumer contacts per month.
- This equals 36,000 consumer contacts per year.

Howard County's population is approximately 225,000. Therefore, this program has the potential to present EMS-EHAC to approximately 15% of the population per year. Having this portion of the population sworn in as "EHAC deputies" could impact many more family members and friends.

When the entire EMS-EHAC program is operational (including the prevention, stratification, and treatment segments), outcome studies can be conducted after a predetermined time. The outcome matrix could include:

- Change in the percentage of deaths from MI
- Change in the percentage of number of MIs
- Change in the percentage of chest pain patients utilizing 911
- Change in the Acute Prevention Index¹⁶
- Change in the time from initial symptom to initiation of therapy

Health-economic analysis

Outcome studies provide only part of the information needed to justify the value of EMS. The second part of the equation is the cost analysis of the medical program.

Saved revenue for health care payers. The GUSTO I trial stated that the average cost for health care of an MI patient treated with thrombolytic drugs is \$200,000.¹⁵ Therefore, if there is a reduction of ten MIs in a geographical area, the health care costs are reduced by \$2,000,000. In areas where EMS has a direct link to managed care, this is a direct cost savings (\$2,000,000 could pay the annual salaries and benefits of 40 to 50 paramedics). The cost of an early symptom-based EMS-EHAC program is negligible compared to the expense of a myocardial infarction.

Saved revenue for the consumer. For every heart attack prevented, a consumer continues to participate as an active

citizen. Assuming that the average salary is \$30,000 per each reduced heart attack, and the EMS-EHAC program can reduce the number of heart attacks by 10, this saves \$300,000 in personal earnings for these families.

Saved revenue for the local tax base. For every heart attack prevented, a consumer also continues to participate as a taxpayer. Therefore, using the same assumptions, a \$30,000 taxable income paying 4% local tax and \$2,000 property tax would result in a savings of \$32,000 in revenue for the local tax base, if EMS were able to prevent ten heart attacks.

SECTION II Stratification

Introduction

Emergency heart attack care is an educational prevention program designed to significantly reduce the occurrence and damage of heart attacks. EHAC is based on teaching people to spot the mild intermittent warning signs of an oncoming heart attack and encouraging individuals to seek prompt medical attention.

Heart attacks have beginnings, or prodromal stages, with recognizable symptoms in most patients.¹² By recognizing these symptoms, patients could ensure that they receive thrombolytic drugs early in their heart attack and possibly avoid severe, permanent myocardial damage. In addition, acute prevention of heart attacks can take place much more effectively at the prodromal stage than during the attack stage. Thus, patients may increase their chances of maintaining a functioning heart by seeking medical attention upon becoming even mildly symptomatic.¹²

What role does EMS have in the continuity of patient care?

By exploring and determining the answers to the following questions, it will be obvious that EMS plays an integral part not only in EHAC, but also in all areas where prehospital patient care has an influence. These areas of influence include patient care, managed care, health care cost containment, effective utilization of existing resources, and increasing patient life span.

Questions to consider:

- Can we identify cardiac ischemia in patients who have atypical presentations?
- Can we better select patients who need the coronary care unit (CCU)?
- Can we shorten the time to rule out MI, thus reducing system/patient costs, more effectively utilizing EMS resources, and reducing hospital admissions and average length of stay?
- Can we decrease the morbidity and mortality of unstable angina?

- Can we better treat acute MI?

EHAC: a two-part community education program of recognition and response

EHAC is a two-part community education program. By recognizing the signs of cardiac ischemia, the patient, family member, or other bystander initiates an immediate response by seeking assistance. This is different from CPR, which is based on training an individual to react to cardiac arrest.

There are certain early warning signs of a heart attack (Table 2). Those warning signs and the following points are stressed to EHAC audiences:

- Heart attacks have beginnings as intermittent and mild chest symptoms.
- Half of all heart attack patients recall having one or more of these symptoms prior to the onset of their severe heart attack pain.
- The chest symptoms generally worsen with increased exertion or exercise and usually improve with rest.
- These symptoms can persist for several hours or days prior to the heart attack.
- They occur more frequently and increase as time passes.

It is well documented that patients experiencing acute chest discomfort often deny these symptoms,¹⁷⁻¹⁹ thus delaying treatment. By introducing the patient to definitive out-of-hospital ALS, the patient's chances for survival are improved.²⁰

Therefore, the response to these symptoms should be to call 911. EHAC videotapes currently available in Blockbuster Video stores do not mention 911 as the primary, appropriate response. Calling 911 is the national standard for emergency assistance.^{18,21} The public should be encouraged to utilize resources with which they are already familiar.

The public should NOT be encouraged to decide whether to go to the emergency department (ED) by 911 or private vehicle. This decision is best left to professionals who are trained and educated to determine the appropriate mechanisms of treatment and transportation. Several EMS agencies nationwide have begun Expanded Scope of Practice programs that allow the paramedic to determine the best course of action to take with the patient, including referrals to private physicians when appropriate. In these systems, 911 is activated for anyone in need (or a perception of need) and a paramedic responds and acts according to established guidelines and protocol.

The emergency department chest pain center

A chest pain center (CPC) is an expansion of the hospital's heart station into the emergency department. A CPC serves

TABLE VI. The EMS bridge

- Involves training EMS personnel in the concepts of EHAC
- Reduces "door-to-needle" time for thrombolytic, PTCA, or sestamibi treatment
- Utilizes prehospital 12-lead ECG testing and diagnosis

as a command post for quickly identifying and attending to patients experiencing, or at risk to experience, a heart attack. In compliance with American College of Emergency Physicians' (ACEP) Guidelines, a CPC includes these seven features and functions:

1. Fast track for patients with acute myocardial infarction.
2. Observation area for patients with acute myocardial ischemia.
3. Detection program to target and educate patients with coronary artery disease (based on a positive stress test) and patients with increased factors (based on history and blood tests).
4. Education outreach program, in conjunction with the hospital's rehabilitation department, to communicate to the public that warning signs precede heart attacks.
5. Medical educational programs involving continuous quality improvement (CQI), Health Care Financing Administration (HCFA) MI indicators, National Heart Attack Alert Program (NHAAP) recommendations, etc. The goal is the relentless pursuit of teamwork for maximal benefit to the patient, the hospital, and society.
6. Proper staffing ratio of critical care nurses, as well as critical care physicians with a continuous retraining program for heart attack management.
7. Appropriate technology that is timely and available 24 hours, with prompt backup interpretation. These include enzymes (CPK/MB, myoglobin, and Troponin), ST monitoring, technetium sestamibi, nuclear testing, echocardiography and stress testing.

EHAC's goal is to have a CPC in every U.S. hospital, so as to provide the penetration and hospital connection necessary to reach patients with acute myocardial infarction and acute myocardial ischemia early enough to intervene and introduce preventive practices. The strategy is to prevent disease and promote health.²²⁻²⁵

EMS and EHAC: a sample case

A 57-year-old man complains of indigestion to his wife. She observes him to be in moderate discomfort with mild shortness of breath. His wife received EHAC education when he was discharged from the hospital last month after unstable angina. She recognizes the possible signs of another heart attack and calls 911.

Fire department first responders arrive quickly and apply oxygen, obtain a detailed medical history, and record vital signs. EMS arrives and obtains a 12-lead electrocardiogram (ECG). Non-specific ST changes are noted when compared to

the patient's baseline copy. Advanced cardiac life support (ACLS) intervention is administered prior to, and during, transport. The paramedic uses the CPC's triage protocol and advises the ED of a level-three chest pain patient (this triage system is discussed later in this article).

Upon arrival at the CPC, the patient is quickly reassessed. Since his discomfort was relieved prior to transport and has not returned, the patient is quickly moved to nuclear medicine for sestamibi testing. The patient is admitted to the critical care unit (CCU) for 24 hours, then to a step-down unit before being discharged.

This sample case demonstrates several benefits of EHAC education:

- reduced myocardial damage
- reduced risk of the "missed MI"
- reduced health care costs
- decreased length of hospital stay
- maintaining the patient's quality of life
- increased life span of the patient

The EMS bridge concept (Table 6)

Dr. James Espinosa, et al., of Overlook Hospital in Summit, New Jersey, promotes the concept of an EMS bridge between the community and the emergency department. In the August, 1996, issue of *Clinician*, they write, "We see the EMS as a bridge to the community, and as a way to focus our energies on reducing prehospital time to thrombolytic treatment. EMS personnel, as natural ambassadors to the community, represent a bridge from the ED to the community, and from the community to the ED. The EMS bridge concept will involve increased training of EMS personnel, at all levels, in the core concepts of EHAC, the 'open artery hypothesis,' and the immense value of a prehospital 12-lead ECG."

The article concludes by expressing the hospital's perception of EMS-EHAC: "We see the EMS bridge as our next step. It satisfies our need to do more, and to reduce time from the onset of symptoms to the activation of the emergency medical system. At the same time, we will create the infrastructure needed for meaningful and lasting behavioral change in the community."¹⁴

As the EMS Scope of Practice continues to expand, providers will be spending more time with members of their community in non-emergency settings. This provides a wonderful opportunity to promote EHAC to the public, especially to residents of medically underserved areas.

From another perspective, the EMS bridge serves as a conduit for relaying patient information, conducting continuing education, and training between the ED and EMS. This conduit works in two directions in systems that promote mutual respect and work toward a common goal.

Indeed, at Summit Hospital, the door-to-needle time for acute MI patients needing thrombolysis was reduced from 75 minutes to 16 minutes,²⁴ with anecdotal evidence suggesting occasional situations of 0 minutes.

Rapid transport: the golden hour not just for trauma anymore

What is the appropriateness of rapid patient transport? Traditionally, rapid transport has been associated with trauma and the golden hour. But, if we are going to reduce myocardial damage by getting the patient to a CPC within the first hour from the onset of symptoms, then we must consider these patients for rapid transport as well.

The decision to transport rapidly is made by the paramedic who must constantly assess the risk/benefit ratio of rapid transport to the ED versus the risk/benefit of remaining on scene and providing treatment. Other factors to be considered include time involved with packaging the patient for transport, distance to the ED, weather conditions, type of transportation (ground vs air), and others.

How often is Priority 1 applied to a patient with stroke symptoms? How many such patients are transported with that same urgency? A golden hour concept has been introduced regarding acute stroke patients. It is referred to as a "brain attack."^{26,27} EMS systems and dispatch centers need to revise their protocols in this regard. Clausen Dispatch Cards (and other advanced medical protocol dispatch systems) still calling for an urgent or non-urgent response to these patients need revision as well.

ACC/AHA practice guidelines Prehospital issues—recommendations

The ACC/AHA Task Force on Practice Guidelines specifically addresses prehospital issues and makes recommendations for promoting rapid identification and treatment of patients with acute MI.²⁸ These recommendations are defined and prioritized by classes. The comments following each recommendation are those of the author.

Class I

Class I recommendations are those that will have a definite benefit to the acute MI patient. These are:

1. Availability of 911 access.

911 access is available to 80% of the U. S. population. The federal government has spent millions of dollars building a 911 telecommunications network throughout the country. Only the most rural and isolated areas are without this universal access to emergency assistance. As mentioned earlier, 911 is the primary appropriate response to any emergency need. This message must continue to be promoted.

2. Availability of an EMS system staffed by persons trained to treat cardiac arrest with defibrillation if indicated and to triage patients with ischemic-type chest discomfort.

Prehospital (or out-of-hospital) ALS is available from a number of different agencies and system types. These are some examples:

EMS/fire departments. Most fire departments require their firefighters to be certified as EMT-Bs. This is also required for firefighters certified to the National Professional Qualifications standard.²⁹ Nationally, fire department responses have shifted from fire-related calls to requests for EMS assistance. Many of these agencies have put at least one paramedic with ALS equipment on their fire apparatus. These ALS engines typically arrive faster than an ambulance and provide for an advanced first response.

Hospital-based EMS. Hospital-based EMS will become more prevalent as managed care expands. There is money to be made in patient transportation, but not from 911. Managed care will end up providing EMS as the cost of doing business in their communities.

Health departments. Health departments are not typically associated with EMS but play a vital role in the regulation and licensure of these services. Home health nursing is often provided through this or other social service agencies. A registered nurse or other health care professional is usually the primary provider for these agencies, though rarely in an ALS capacity.

Private sector. The private sector is another key player in EMS. Like managed care facilities, the private sector is in the business of making money and this is accomplished by transporting patients. Look for the largest private sector ambulance services to begin purchasing insurance companies. This will ensure a source of revenue since they will transport their own members. This is where Expanded Scope of Practice needs to be closely monitored to ensure that patients are receiving care based on their needs and not that of the private service.

Volunteers. EMS and fire volunteers are still the unsung heroes in our communities but are disappearing quickly as increased training and continuing education requirements put too much of a burden on people's time. But remember, whether one is a volunteer provider or a career provider, they are still professionals.

Finally, this recommendation calls for a sense of urgency on the part of EMS personnel in managing patients with ischemic-type chest discomfort. The goal is to have a trained professional interacting with the patient within five minutes of the call for help.

Class IIa

Class IIa are those recommendations that will probably benefit the patient. These are:

1. Availability of a first-responder defibrillation program in a tiered response system.

There are many types of first-responder programs. Fire departments, EMS, police departments, rescue squads, coworkers, bystanders, and others can all receive training in the use of the automatic external defibrillator (AED). Not only are these devices effective and safe,³⁰⁻³⁴ they are also easy to use with a minimum of training. These devices are becoming more commonplace and can often be found in shopping malls, on airplanes, in hospitals, clinics, and sports complexes. Don't be surprised if someday an AED is as accessible as a fire extinguisher. Recently, the first AED was approved for use on passenger airplanes.

2. Health care providers educate patients/families about signs and symptoms of acute MI, accessing EMS, and medications.

This recommendation speaks directly to the concepts of EHAC. Community education and training programs such as EHAC and CPR stress a focused response. It is said that EHAC is for live patients and CPR for dead ones. This is because EHAC is proactive. Thus, EHAC can potentially reduce the need for CPR.

Patient action plans are currently a part of a hospital's discharge planning process. Patients should be educated about the use of their medications and what they do. Aspirin and nitroglycerin are two examples. Patients should also be instructed on how to access EMS. Where 911 is not available, telephone stickers need to be provided to the patient and their families. EMS Expanded Scope of Practice will deal with this type of planning as well.

All patients with a cardiac history should be given a copy of their 12-lead ECG as a baseline for EMS and ED to use in the evaluation of the patient. A listing of the patient's current medications should be attached to the ECG as well. Frequently treatment is delayed without this information.

Class IIb

Class IIb recommendations are those that have a possible benefit to the patient. These are:

1. 12-lead telemetry.

Prehospital 12-lead ECG testing is becoming a standard of care performed by highly skilled and educated ALS providers. There are two types of prehospital ECG programs.

Off-line medical control. Paramedics perform 12-lead ECGs and other patient care via indirect physician control per system protocols and procedures.

On-line medical control. Direct control of patient care is provided to paramedics via communications between medical control and field personnel.

While managing the patient's chest discomfort, the paramedic performs a 12-lead ECG. If the patient is diagnosed as

experiencing an acute MI, the paramedic evaluates the patient for thrombolytic therapy. This is accomplished by using a checklist such as the one published by the AHA (Table 7). Then the 12-lead ECG and the results of the evaluation are communicated to the ED during transport. It is the delivery of this information to the ED and its appropriate capture and utilization that reduce door-to-needle time.

In addition to rapidly identifying acute MI, prehospital 12-lead ECG can be used to diagnose angina patients who are candidates for sestamibi testing. This cardiac-specific nuclear medicine, like thrombolytics, requires preparation prior to use. Later in this article we will discuss how to integrate this ability into current EMS practices.

2. Prehospital thrombolysis in special circumstances (e.g., transport time greater than 90 minutes).

It seems there are always discussions about the appropriateness and efficacy of prehospital thrombolytic therapy. The MITI-1 stratification trial performed in Seattle in the 1980s attempted to address these concerns.³⁵ The objective of the trial was to determine

TABLE 7. Chest pain checklist for use by EMT/Paramedic for diagnosis of acute myocardial infarction and thrombolytic therapy screening

Check each finding below. If all [yes] boxes are checked and ECG indicates ST elevation or new BBB, reperfusion therapy with thrombolysis or primary PTCA may be indicated. Thrombolysis is generally not indicated unless all [yes] boxes are checked and BP <180/110 mm Hg.

	Yes	No
Ongoing chest discomfort (>20 min and <12 h)	_____	_____
Oriented, can cooperate	_____	_____
Age >35 y (>40 if female)	_____	_____
History of stroke or TIA	_____	_____
Known bleeding disorder	_____	_____
Active internal bleeding in past 2 weeks	_____	_____
Surgery or trauma in past 2 weeks	_____	_____
Terminal illness	_____	_____
Jaundice, hepatitis, kidney failure	_____	_____
Use of anticoagulants	_____	_____

Systolic/diastolic blood pressure

- Right arm: _____ / _____
- Left arm: _____ / _____

	Yes	No
ECG done	_____	_____

*High-risk profile**

	Yes	No
Heart rate >100 bpm	_____	_____
BP <100 mm Hg	_____	_____
Pulmonary edema (rales greater than one half way up)	_____	_____
Shock	_____	_____

**Transport to hospital capable of angiography and revascularization if needed.*

Pain began	AM/PM
Arrival time	AM/PM
Begin transport	AM/PM
Hospital arrival	AM/PM

EMT indicates emergency medical technician; ECG, electrocardiogram; BBB, bundle branch block; PTCA, percutaneous transluminal coronary angioplasty; BP, blood pressure; TIA, transient ischemic attack. Adapted from the Seattle/King County EMS Medical Record.

Contraindications and Cautions for Thrombolytic Use in Myocardial Infarction*

Contraindications

- Previous hemorrhagic stroke at any time; other strokes or cerebrovascular events within 1 year
- Known intracranial neoplasm
- Active internal bleeding (does not include menses)
- Suspected aortic dissection

Cautions/relative contraindications

- Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)+
- History of prior cerebrovascular accident or known intracerebral pathology not covered in contraindications
- Current use of anticoagulants in therapeutic doses (INR >2-3); known bleeding diathesis
- Recent trauma (within 2-4 weeks), including head trauma or traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Noncompressible vascular punctures
- Recent (within 2-4 weeks) internal bleeding
- For streptokinase/anistreplase: prior exposure (especially within 5 d to 2 y) or prior allergic reaction
- Pregnancy
- Active peptic ulcer
- History of chronic severe hypertension

INR indicates International Normalized Ratio; CPR, cardiopulmonary resuscitation. *Viewed as advisory for clinical decision making and may not be all-inclusive or definitive. +Could be an absolute contraindication in low-risk patients with myocardial infarction (see text).

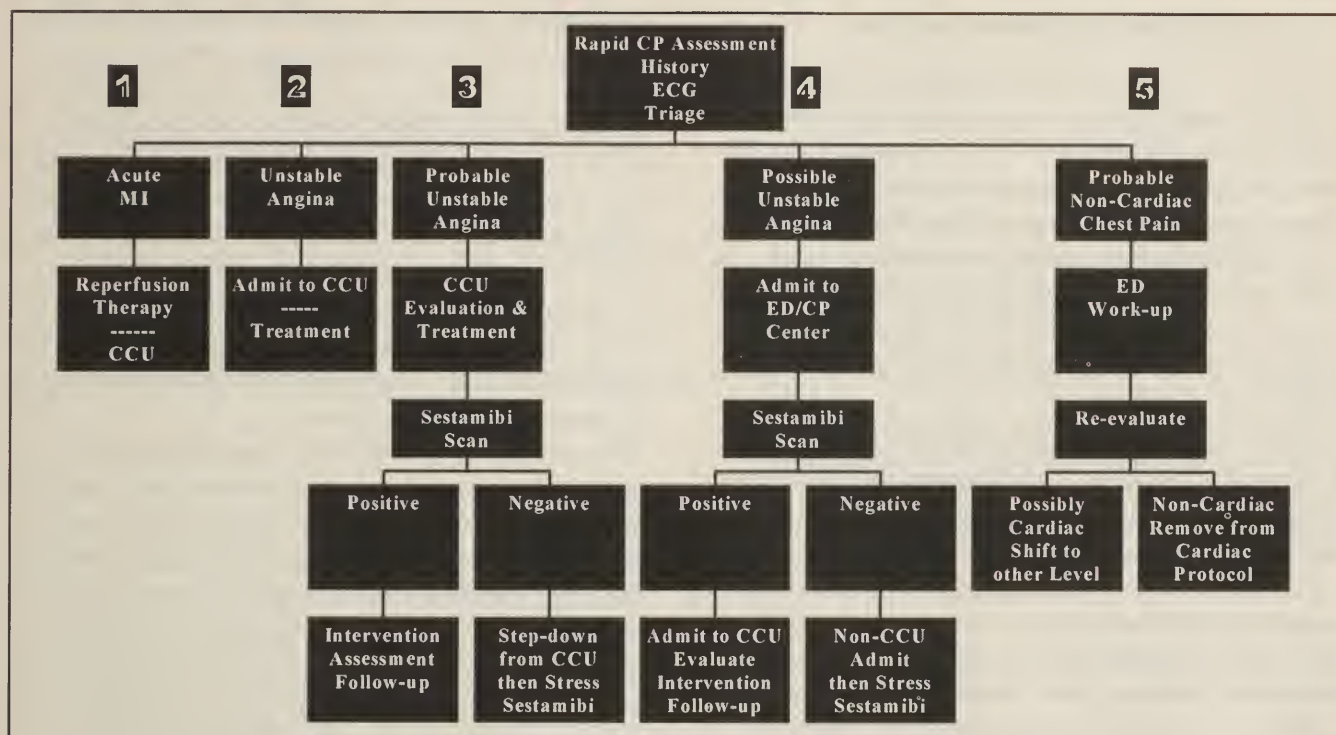


Figure 1. ED chest pain protocol levels of chest pain assignment

feasibility of prehospital thrombolytics by paramedics in patients with acute MI.

The study concluded that paramedics could identify candidates with the use of a checklist and 12-lead ECG, and as a result could benefit patient outcome by saving time and possibly reducing the complications and extent of infarction. The new practice guidelines adopt these conclusions as being of possible benefit to the patient in special circumstances. These special circumstances are dependent on transportation times.

As with all actions and considerations, there are pro and cons. In regard to prehospital thrombolysis, the main advantage is:

- 17% relative improvement in patient outcome especially when therapy is given 60 to 90 minutes earlier than in the hospital.²⁸

Disadvantages include:

- a small percentage of patients eligible for thrombolysis; selection of patients can be difficult (even for physicians), and
- various medical, legal and economic implications of thrombolysis.²⁸

When the door-to-needle time is not improved in a system that does not offer prehospital thrombolysis, there may be benefit of doing it in the field. However, a general national policy regarding prehospital thrombolysis is not currently advocated by the ACC/AHA except in special settings.

Retavase

Retavase™ is a new thrombolytic drug that was cleared for use by the U.S. Food and Drug Administration in October 1996. It has been proven to be safe and effective in reducing mortality. It is administered in a two shot dose, (an immediate bolus and a repeat bolus 30 minutes later), rather than by continuous intravenous infusion. It does not require refrigeration and thus has the potential for prehospital administration. Prehospital studies need to be performed to determine the efficacy of adding this drug to our arsenal.

ACC/AHA practice guidelines: prehospital issues—selected data and interpretations

There are 900,000 cases of acute MI annually in the United States.²⁸ This figure is greater than the population of the city of Baltimore. Almost one in four of these people die,²⁸ and half of the patients who die will do so within the first hour of experiencing symptoms.²⁸

This is why EHAC needs to be advocated and taught with the same sense of urgency as CPR, anti-smoking campaigns and wellness programs. We have the ability to affect the lives of more than just these 112,000 patients. Health care costs will be reduced, people will live longer, will remain in the workforce longer, and retain their quality of life.

Integrating a 12-lead ECG program into a mechanism that would also benefit patients experiencing angina is a natural progression for EMS and our role in the continuity of patient care. The chest pain protocols at the Medical College of Virginia are shown in **Figure 1**. Once the paramedic has performed appropriate patient testing and evaluation, the patient can be assigned an acuity level. This acuity level can then be communicated to the ED and preparations for the patient can be made prior to arriving at the chest pain center.

Level 1 patients are diagnosed with an acute MI and are at high risk of mortality. Rapid transport, ACLS intervention and thrombolytic therapy are indicated with an admission to the CCU.

Level 2 patients are definitely experiencing unstable angina. These patients are also at high risk of mortality and need rapid transportation, ACLS intervention, and comprehensive evaluation and treatment in the chest pain center.

Level 3 and 4 patients are at moderate risk of mortality. Distinguishing between these two acuity levels is not critical in the pre-hospital setting. The decision to transport rapidly is made by the paramedic based on patient presentation and other factors to be considered by the paramedic.

Level 5 patients are diagnosed as experiencing noncardiac symptoms after the paramedic elicits a detailed patient history and examination including a 12-lead ECG. These patients are at low risk of mortality and are treated and transported as necessary and appropriate. Expanded Scope of Practice will benefit the patient and health care system for these patients.

Summary

EMS-EHAC is a complete chest pain program that includes prevention, stratification, and treatment strategies. The prevention segment of EMS-EHAC, if executed properly, could potentially have the greatest impact in reducing cardiac death and myocardial infarction by educating health care providers and the public to recognize and react to the early, subtle prodromal symptoms that occur in 50% of the patients who have infarctions. The Class IIb section regarding patient and family education of the ischemic patient in the ACC/ACA guidelines is also satisfied. These guidelines from the ACC/AHA have a stronger level of recommendation than the 12-lead and thrombolytic programs that are currently in use or under development by many EMS agencies. EMS-EHAC is a program that is designed to continually promote the symptom recognition

message, and is associated with minimal design and implementation costs. The EHAC messages are very similar to stroke recognition programs, and the patient type is quite similar. EMS-EHAC programs have the potential to not only assist in the early recognition of chest pain, but signs of a "brain attack." New therapies for the stroke patient including thrombolytics and neuroprotective agents have similar potentials in morbidity and mortality reduction.

The public can benefit from EMS-EHAC due to the increased awareness of chest pain and its inherent risks, the quality of their EMS system, and the enhanced relationship with their local health business.

The value of a product is measured as the sum of its quality and its cost. Advanced life support EMS will not be immune from health care scrutiny, therefore outcome studies must be developed to demonstrate superiority versus competing modalities (ALS vs enhanced BLS), along with health economic analyses. The costs analysis should not be based on structure or outcome of the EMS system, but rely on the outcome data that could demonstrate the upfront investment in EMS translate to an overall cost saving for health care payers and consumer. Marketing and branding of EMS services should not be ignored. The value of EMS must be communicated to the health care payers, general public, and government agencies to support their investment in the EMS structure.

The prevention, stratification, and treatment plan described in this article is the culmination of the best practices in chest pain care today. Successful implementation of this plan could result in the ultimate goal of the EHAC program; the removal of heart disease as the number one killer.

We have shown you that there are many roles for EMS and their providers in the delivery of a team approach to patient care. The EHAC message is a natural one for EMS and Fire agencies to market to their communities. Just as treatment modalities are introduced and integrated into hospital-based health care systems, these same issues need to be considered for prehospital application as well.

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Emergency Medical Service system evaluation and planning strategies for prehospital chest pain in Howard County, Maryland

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ABSTRACT: *Patients experiencing ischemic chest pain represent one of the most common emergencies in prehospital emergency medical service (EMS) systems. Recent national guidelines for emergency department and EMS care of chest pain and acute myocardial infarction (AMI) patients have quantified standards for time to evaluation and treatment. Prehospital EMS systems and hospitals will need to change their processes of care for chest pain patients to meet or exceed these national guidelines. In addition, the EMS system and the hospital will need to work more closely and seamlessly to integrate chest pain care for the maximum benefit of the patient.*

The Howard County Department of Fire and Rescue Services (DFRS) recognized the need for change in its EMS system. Its unique approach involved chartering a multidisciplinary task force to assess the current EMS system. The task force analyzed the literature to decide evidence-based standards for EMS system performance, and evaluated public and private EMS systems that possessed state-of-the-art processes for providing patient care. Based upon the information gathered and considering qualities essential to future performance, the task force made recommendations for changes to Howard County's EMS system.

This study reports the task force recommendations and describes those implemented thus far. Changes to the EMS system are quantified, descriptive data from the system are reported, and future goals are presented.

Introduction

The spectrum of ischemic heart disease spans a continuum from asymptomatic disease to acute myocardial infarction (AMI) to cardiac arrest. Prehospital and hospital care is focused on rapid identification and treatment of candidates most able to benefit from interventions; these interventions are designed to decrease ischemia and infarction, and to preserve left ventricular function.

In the United States, approximately one million people annually experience AMI. More than 50% of the AMI patients die in the out-of-hospital setting.¹ In Maryland, there are approximately 30,000 AMI patients annually, with 15,000 deaths estimated per year. Interventions in cardiac arrest and AMI which affect survival (such as defibrillation and reperfusion) are time dependent; the greatest benefit is realized from the most rapid treatment.

Prehospital and hospital care for ischemic chest pain patients has changed dramatically in the past decade. The American Heart Association has identified factors critical to successful resuscitation from sudden cardiac death: the chain of survival. The four interdependent factors in this process include the following links: 1) early access to care (or 911), 2) early cardiopulmonary resuscitation (CPR), 3) early defibrillation, and 4) early advanced life support (ALS) care.² Interventions including reperfusion therapy (i.e., thrombolytic therapy and percutaneous transluminal coronary angioplasty [PTCA]) have improved outcomes for patients with AMI while defining time to treatment as a critical factor. Chest pain evaluation centers have provided more accurate identification of ischemic chest pain patients. Guidelines from the American Heart Association³ and the National Institutes of Health (National Heart, Lung and Blood Institute, National Heart Attack Alert Program)⁴ have established and refined standards for time to treatment in AMI and defined the effectiveness of other interventions. Prehospital 12-lead electrocardiogram (ECG) acquisition has been shown to decrease emergency department time to thrombolytic therapy.^{5,6,7}

Evaluation

Over the past decade the percentage of fire department emergency medical service (EMS) calls, as a function of all fire departments calls has been increasing nationally, with the majority of total responses coming from EMS calls. In Howard County, Maryland, during 1994, more than 70% of total department responses involved care for patients, rescue, and extrication.⁸ Focused upon improving prehospital patient care, the director of the Howard County Department of Fire and Rescue Services (DFRS) commissioned a task force to study EMS services in October 1994.

The task force was conceived to be multidisciplinary and community based; it included civic and business leaders, citizens, physician representatives from the medical community and the hospital and representatives from volunteer and career fire services.

The group conducted searches of medical and EMS literature to define standards for EMS system attributes and performance. In addition, they studied both fire service and public utility EMS systems to evaluate the effect of different EMS models on the delivery of prehospital care.

In March 1995, the EMS task force produced its final report, including 33 recommendations for making the EMS system of the Howard County DFRS a "pre-eminent public sector provider of EMS in the state of Maryland." The recommendations that impact patient care can be grouped into these categories: the chain of survival, provider certification, response times, a continuous improvement program, community awareness/education, medical direction, data collection, and preventative/well programs (Table 1).

Demographics and population distribution

Howard County is the sixth most populous jurisdiction in Maryland. The estimated population in 1995 was 218,400—4.33% of Maryland's population. The population of adults aged 60 and older is estimated to be 8.0% of the total county population. Howard County is one of three counties expected to have the greatest increase in adults greater than 60 years old by the year 2020 (projected at a 257.9% increase).⁹ It is in this segment of the population that heart disease most frequently manifests itself.

The total area of the county is 252 square miles. Densely populated areas like Columbia and Ellicott City comprise 40.5% and 27.1% of the population, respectively. Geography and population distribution have specific implications for the time dependent prehospital processes and interventions used in cases of sudden cardiac death and chest pain.

In 1995, the frequency of cardiac arrest in Howard County was 232, the sixth most frequent rate among jurisdictions in Maryland. From 1992 through 1994, heart disease and stroke matched cancer as the leading cause of death in this county.

Implementation

The task force's final report was published March 29, 1995. Progress in selected task force recommendations will be described in the following content areas.

Provider certification

Basic life support. Emergency medical technician-basic (EMT-B) has been established as the minimum standard of basic life support (BLS) care. Important interventions for

chest pain patients such as sublingual nitroglycerin and automated external defibrillation (AED) are additions to the scope of practice of EMT-Bs which were not available in the emergency medical technician-ambulance (EMT-A) curriculum. Working within the state's framework for recertification, 24-hour courses to educate EMT-A providers to become EMT-Bs (bridge classes) have begun. Based on the certification expiration date of the EMT-As, bridge classes to EMT-B will be complete by June 30, 2000. There have been seven EMT-B bridge classes since July 1996, the date that Maryland initiated EMT-B as a state certification level. Thus far, bridge classes have educated 92 providers. As a result, 44 additional providers in the system have the enhanced capabilities of AED, and can aid the patient with sublingual nitroglycerin administration.

Advanced life support: The minimum level of ALS care has been established to be emergency medical technician-paramedic (EMT-P). Prior to release of the task force's final recommendations there were 27 career ALS providers in the system. Provider deployment at that time was via three "chase cars" — ALS ambulances staffed with paramedics which would rove the county (not assigned to specific fire stations) responding to ALS calls. The ability of the chase cars to roam the county provided flexibility though the limited number of units can sacrifice response time in critical processes such as sudden cardiac death and chest pain. Since publication of the task force recommendations there has been one cardiac rescue technician (CRT) to EMT-P bridge class; this produced an additional 20 career and volunteer paramedics, and 20 paramedics have been hired as firefighter trainees. This tripling of paramedics has allowed staffing of the majority of stations with two paramedics — one on the ambulance and one on the fire engine. This new deployment plan provides a primary paramedic response for both ambulances and engines, which makes reaching response time goals feasible.

Response times

Sudden cardiac death and acute myocardial infarction are prehospital conditions dependent upon timely interventions for optimal outcomes. Specific review of the epidemiology and treatment of sudden cardiac death defines the issue in more

TABLE 1. EMS task force recommendations

Chain of survival

- All 911 personnel undergo priority dispatch training
- Institute a comprehensive program for community CPR training
- Place automated external defibrillators (AEDs) on all BLS response units and train EMT-A personnel in their use
- Establish the minimum level of advanced life support of patient care as EMT—Paramedic

Provider certification

- EMT-basic is established as the minimum level of basic life support (BLS) care
- EMT - paramedic is established as the minimum level of advanced life support (ALS) care

Response times

- 911 call receipt to BLS provider at patient side: 10 minutes
- 911 call receipt to ALS provider at patient side: 12 minutes

Continuous improvement program

- Establish an ALS continuous quality improvement program
- Establish clinical criteria for continuous system evaluation and development

Community awareness/education

- Develop and present a comprehensive program for public education and awareness
- Create a public service unit to be used as a community education unit

Medical direction

- Establish a position for a compensated medical director, certified in emergency medicine with broad experience in EMS

Data collection

- Obtain an appropriate data collection, management, and analysis system for operational records

Preventive/well programs

- EMS personnel, in concert with the medical director, should become involved in preventive and community-based wellness programs

detail. Victims of sudden nontraumatic cardiac arrest are found to be in ventricular fibrillation 80% to 90% of the time.¹⁰ The treatment for ventricular fibrillation is defibrillation. Studies of post-MI patients who sustain sudden cardiac death in the cardiac rehabilitation setting reveal that, with rapid defibrillation, return of circulation occurs in 80% to 90% of these victims.¹¹ When the victims of sudden cardiac death sustain their cardiac arrest in the prehospital setting and receive care in the most successful systems — systems that report CPR and defibrillation within four minutes and ALS care within

Figure 1a. Medical incident report (MIR) form. Figure 1b. Medical incident report (MIR) form.

eight minutes — the maximum reported return of circulation falls in the range of 30% to 40%. Thus EMS system planning must take into account the relationship of time (the interval from 911 call to defibrillation) and survival.

The prehospital approach to chest pain is also a time-dependent function. Guidelines from the American Heart Association (AHA)¹² and the National Institutes of Health⁴ have established that prehospital and emergency department systems must be in place to accomplish a 12-lead ECG within ten minutes, and a door-to-needle time of less than 30 minutes for chest pain patients. It has been demonstrated that prehospital 12-lead ECGs can decrease door-to-data time, and ultimately door-to-drug time, in AMI patients.^{5,6,7}

Typically reported in the literature is the 911 vehicle dispatch-to-scene arrival interval; this does not account for the time it takes for the provider to arrive at the patient's side to provide care.¹³ According to the literature, guidelines for EMS 911 to arrival-at-scene intervals are eight minutes for ALS and four minutes for BLS. The task force recognized the importance of measuring the interval of 911 call to arrival at patient as a more accurate measure of time to treatment. There is no data linking this interval (911 call to arrival at patient) to survival of cardiac arrest, or to outcome for chest pain patients; therefore, the task force established intervals of ten minutes for BLS, and 12 minutes for ALS. These newly established response time intervals will be linked to data on the outcome of cardiac arrest to determine the validity of these intervals, and to establish thresholds for response.

Continuous improvement program

Quality improvement has particular importance for the evaluation and planning of the EMS response to chest pain

and sudden cardiac death. The tenets of a quality improvement program include these steps: 1) define the process, 2) determine root causes of delays or problems, 3) collect data, 4) analyze the process based on the data, 5) make changes based on the data, and 6) re-analyze the process after changes are implemented.

A quality improvement program has been established in the Howard County DFRS; the medical director, the EMS program manager, and the medical duty officers (EMS supervisors) were in-

cluded. The group has begun evaluating prehospital processes for chest pain and sudden cardiac death. A group of knowledgeable participants providing feedback through data is critical to analysis of this process, to the recommendation of changes, and to EMS system improvement. This group can then re-evaluate the process after implementation of changes, and can modify the system based on objective data.

Community education/awareness

The second link in the AHA's chain of survival is early CPR. The community is the first provider of emergency cardiac care; bystander CPR is a critical link to providing less-than-normal ventilation and perfusion until EMS providers can respond with an AED and advanced cardiac life support (ACLS). Recognizing the importance of citizen CPR and the mission of the Howard County DFRS to educate the public, a citizen CPR program was initiated in 1995. The CPR hotline allows citizens to call the DFRS 24 hours a day to obtain information about the program, register for a course, or leave a message requesting information. In 1996, this citizen CPR program educated over 1,500 citizens. In addition to the actual procedure of CPR, the course provides a forum to educate citizens about symptom recognition, risk factor assessment, and lifestyle modification. In May 1997, the state of Maryland and the Maryland Institute of Emergency Medical Services Systems (MIEMSS) honored the Howard County DFRS for its citizen CPR program by presenting them with the EMS program award.

Medical direction

The EMS task force used MIEMSS, American Society for Testing and Materials descriptions, and literature from the evidence-based medicine search to define the duties of the

A R R E S T	CPR BEFORE HCFR ARRIVAL: <input type="radio"/> Police <input type="radio"/> Bystander <input type="radio"/> Dispatch Asst. <input type="radio"/> None <input type="radio"/> Unknown <input type="radio"/> N/A <input type="radio"/> Other							1st HCFR CPR <input type="radio"/> ALS <input type="radio"/> BLS		
	FIRST CARDIAC ARREST RHYTHM / STATE <input type="radio"/> VF <input type="radio"/> V Tach <input type="radio"/> Asystole <input type="radio"/> Not Monitored, BLS <input type="radio"/> PEA <input type="radio"/> Unknown			AED 1ST APPLIED BY <input type="radio"/> EMT <input type="radio"/> MEDIC AED SHOCK BY <input type="radio"/> <input type="radio"/> DEVICE NO. _____		WAS ARREST / COLLAPSE SEEN OR HEARD? <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Unk.		IF WITNESSED, WAS THERE A DELAY TO CALL 911? <input type="radio"/> Yes <input type="radio"/> No <input type="radio"/> Unk. <input type="radio"/> N/A		
								<input type="radio"/> CARDIAC ARREST WITNESSED AFTER ARRIVAL OF 1st HCFR UNIT		
	C/A WITNESS (Mark all that apply) <input type="radio"/> Family <input type="radio"/> Bystander <input type="radio"/> Co-worker <input type="radio"/> Supervised medical <input type="radio"/> Other							<input type="radio"/> IMPLANTED AICD		
							WAS WITNESSED CARDIAC ARREST ECG MONITORED? <input type="radio"/> Yes <input type="radio"/> No			

Figure 2. Process evaluation.

EMS medical director. From these references a job description was created. The director of the Howard County DFRS convened a multidisciplinary search committee for the medical director position. The search committee used a competitive interview process and objective criteria from the job description to make their recommendation. In May 1996, a part-time medical director was hired.

Over the past year, the medical director has been involved in many task force recommendations. For items associated with the chain of survival the medical director has: 1) developed a course on AED and presented it to instructors and students (to date this course has educated 209 providers, including the 92 providers educated in the EMT-B bridge class; as a result, AEDs have been placed on all BLS response units), 2) participated in a priority dispatch course for emergency medical dispatchers, 3) contributed to planning sessions about the DFRS citizen CPR program with AHA, and 4) evaluated EMT-P candidates prior to clinical patient care and taught initial DFRS training to newly-hired paramedics. The medical director is part of the team which performs the quality improvement analysis on EMS processes.

Data collection

The EMS task force noted that objective data needed to judge EMS performance are unavailable. The patient care record in use at the time of the task force assessment was the statewide Maryland Ambulance Information System (MAIS). The MAIS form collects demographic data, assessment, and treatment information. Though it does collect assessment and treatment information about cardiac arrest, the data collected for chest pain were insufficient to evaluate the assessment and treatment of ischemic chest pain. Following information gathering trips to other EMS systems, Howard County DFRS EMS personnel designed a custom-made form: the medical incident report (MIR). This form collects data essential for the assessment of chest pain care (Figures 1a and 1b). In addition, the MIR was designed for use with form recognition software (Teleform). This feature allows the MIR to be faxed from each fire station to a central data server.

Once successfully received by Teleform, this information is held in the server until it undergoes a quality check by a

verifier. Every MIR also undergoes EMS medical quality improvement review by the EMS supervisor (the medical duty officer). Following verification of MIR, the data fields on the form are translated into tables and data fields for use in a custom-designed Visual FoxPro database. This database is linked to EMS response data (911 call receipt, ambulance dispatch, ambulance arrival at scene, provider arrival at patient side, ambulance departure for hospital, ambulance arrival at hospital, and ambulance back in service). Once data are entered into Visual FoxPro, queries may be written to define the important aspects in the care of the ischemic chest pain patient.

The Physio-Control Life-Pak 300 semi-automatic external defibrillator effectively treats sudden cardiac death by allowing basic EMTs to defibrillate. The Physio-Control Life-Pak 11 monitor/defibrillator/pacer treats a wider range of conditions in patients experiencing ischemic chest pain. It can also record a 12-lead ECG from chest pain patients. Cardiac rhythm data and information on defibrillation timing and energy are recorded by the device. In addition it allows providers (through the use of designated "hot keys") to enter information about dosage and timing of administered ACLS drugs.

Data from both the Life-Pak 300 and the Life-Pak 11 are downloaded to Code-Stat, a cardiac arrest and chest pain descriptive database. This database, written for Microsoft Access, provides reports describing elements of care for chest pain and cardiac arrest. Data from Code-Stat can then be used by Howard County DFRS managers and the medical director to evaluate the process of care for chest pain patients.

Twelve-lead ECG program

Cardiac arrest is one of the few diagnostic categories which has been reported in the literature sufficiently to allow EMS system versus national statistics evaluation. Thus, the process of care for the victim of cardiac arrest is one benchmark upon which to evaluate EMS systems. The MIR was designed to collect data related to cardiac arrest and thus allow process evaluation (Figure 2). System planning is frequently based upon meeting or exceeding guidelines set out by the AHA's chain of survival. Despite intensive planning and implementation, prehospital interventions focused on cardiac arrest have a

maximum reported success rate of approximately 30% to 45% because cardiac arrest is the final condition at the end of the spectrum of cardiovascular disease. Efforts which are focused earlier in the spectrum (for example, those aimed at patients with symptomatic ischemic chest pain) have the potential for greater success because the survival rate is higher.

System improvements that focus on improving the chain of survival are also essential elements in the process of care for symptomatic ischemic chest pain patients. An important focus in the treatment of chest pain patients is identification of candidates for thrombolytic therapy. Prehospital 12-lead ECGs have been shown to decrease ED-to-thrombolytic therapy time.^{5,6,7}

Based upon these facts (and following guidance from MIEMSS and published guidelines)¹⁴ a 12 lead ECG program for ALS providers in Howard County was developed in November and December 1996. The objectives of the course included: educating prehospital providers on their role as the first health care provider for the AMI patient; educating the ALS provider about the clinical presentations of AMI, teaching basic ECG interpretation skills, identifying AMI and thrombolytic therapy candidates on the ECG, defining the process of care for the AMI patient; emphasizing the paramedic role in project objectives and success, and teaching protocol and quality improvement.

At the present time, paramedics receive real-time, on-line medical direction on a rotation basis from five different base hospitals in region III. An essential part of the 12-lead ECG program is real-time medical oversight for ECG interpretation, patient management, and identification of thrombolytic therapy candidates. ECG interpretation and thrombolytic therapy patient evaluation are crucial steps taken by the base station to provide quality improvement of 12-lead ECG patient selection. To accomplish base station quality improvement, the University of Maryland's base station agreed to provide sole medical direction for the 12-lead ECG program. This allows base hospital physician education, quality improvement case review, and

a quality improvement feedback loop to occur at a single base station. All base hospital physicians were educated in a 12-lead ECG base station course.

An important element of the planning process for a prehospital 12-lead ECG diagnostic program is a retrospective analysis of system data regarding chest pain. The purpose of this analysis is to identify the number of chest pain patients evaluated and treated by the system, and report the number of prehospital patients with suspected AMI to allow estimation of the number of AMI patients who are thrombolytic therapy candidates. Following transfer of patient care in the emergency department, the prehospital providers complete the MIR (Figures 1a and 1b). The MIR includes accurate identification of the patient medical problem, as well as a chest pain checklist to document important indications and contraindications to hospital thrombolytic therapy (**Figure 3**). Response, scene, and transport time are also reported to determine baseline values for comparison following implementation of the 12-lead diagnostic program. The completed MIR is faxed to a central fax server where the data is translated to the Visual FoxPro database and becomes available for queries about chest pain demographics, prehospital treatment, and prehospital outcomes.

Data was obtained by a Visual FoxPro database query. The definition of a chest pain patient in the query included a prehospital assessment of chest pain, angina, or suspected MI in patients who received nitroglycerin and who had a systolic blood pressure reading

greater than 90 mm Hg. Using this criteria, 2,226 patients were identified as prehospital chest pain patients between January 1995 and January 1996. The subset of chest pain patients suspected to have AMI were identified by including the prehospital assessment of suspected MI with the nitroglycerin and blood pressure criteria; for the same 12-month period there were 498 patients who met the query for hemodynamically stable patients with suspected MI (**Table 2**).

Time interval data were obtained from the 911 computer aided dispatch system and reported to MIEMSS. Mean response time for chest pain

Pt. treated for Cardiac Chest Pain? ☐ Yes ☐ No

MARK FOR ONGOING HEART PAIN

BP RA /

BP LA /

☐ Systolic is > 80 and < 180

☐ Systolic RA vs. LA is less than 20

☐ Diastolic is < 120

☐ S&S of MI

☐ Pain > 15 min & < 12 hrs

☐ Age (35-74)

☐ ST elevations consistent w/ MI

☐ Oriented, can cooperate

☐ Patient consents

☐ Extensive anterior MI / CV shock

S&S BEGAN									
12-LEAD									
LYSIS ORDER									
LYSIS INIT.									

MARK IF HISTORY OF:

☐ Stroke, seizures, brain surgery

☐ Severe trauma < 2 months

☐ Known bleeding problem

☐ GI / GU Bleed < 12 months

☐ Surgery in < 2 months

☐ Terminal Illness

☐ Tumor, AVM, or aneurysm

☐ Takes warfarin (Coumadin)

☐ Kidney or liver problems

☐ Previous Thrombolytic Rx

Figure 3. Chest pain checklist.

TABLE 2. Retrospective prehospital chest pain data

January 1995 to January 1996

Category	Number
Stable adult chest pain	2,226
Suspected MI	498

(defined as 911 call to emergency scene arrival) was 7.9 minutes. Mean emergency scene time for chest pain patients was 17.0 minutes, while mean transport time was 10.2 minutes (Table 3). Scene time will be closely monitored following implementation of the 12-lead ECG program; the goal is to limit the increase in scene time to no more than five minutes.

The process of prehospital 12-lead ECG acquisition, interpretation, and hospital notification is also important. The process begins with paramedics who identify patients complaining of nontraumatic chest pain. The provider begins care according to the MIEMSS protocol for "chest pain, suspected myocardial infarction" by applying oxygen, attaching the patient to a monitor, and establishing an intravenous line. If the patient does not have resolution of pain with one sublingual nitroglycerin, does have a defined stable rhythm and blood pressure, and meets inclusion criteria, then the ALS provider obtains a 12-lead ECG. Once obtained, the ECG is faxed via cellular modem to: 1) the University of Maryland for 12-lead ECG interpretation and medical direction, and 2) Howard County General Hospital if it is the receiving hospital. If the base hospital physician and the ALS provider agree that there are 12 lead ECG criteria which meet the standards for thrombolytic therapy candidates, then the ALS provider will complete a thrombolytic therapy checklist. If the receiving hospital is any hospital other than Howard County General, the ALS provider will notify the receiving hospital via radio and hand the emergency department team the ECG upon arrival. If paramedics respond to a physician's office for a patient complaining of chest pain, care would be initiated based on the chest pain protocol, a copy of the office ECG would be requested, and the patient would be transported. In general, the paramedics would not acquire a prehospital ECG unless requested by the medical control physician via radio. Quality improvement processes will evaluate prehospital field care for suspected AMI and base hospital medical direction and interpretation of prehospital 12-lead ECGs.

Heart disease mortality

From 1992 to 1994, heart disease and stroke approximated cancer as the leading cause of death in this country. The three-year average deaths per 100,000 (age-adjusted) was 99.5, the second lowest rate in Maryland. This represented a reduction of the U.S. Surgeon General's Healthy People 2000 goal of 100.

TABLE 3. Mean time interval data for prehospital chest pain
January 1995 to January 1996

<u>Interval</u>	<u>Time (min)</u>
911 call to arrival on scene	7.9
Scene arrival to departure	17.0
Scene departure to hospital arrival	10.2

Longitudinally, heart disease mortality has been reduced for each three-year period for the past ten years.⁹

Summary

There has been a paradigm shift in the approach to the patient with ischemic heart disease. Both the AHA's chain of survival and the National

Heart Attack Alert Program's rapid identification and treatment of patients with AMI emphasize the system elements necessary to meet standards of performance in cardiac arrest and AMI for prehospital EMS systems and focuses on evaluating the process of care through a feedback loop.

The Howard County Department of Fire and Rescue Services chartered the EMS task force to evaluate EMS system components, processes, and performance and recommend changes to the system. Currently, many of the recommendations have already been implemented. The result is improved care for chest pain patients, and patients suffering cardiac arrest. All EMTs were educated in an AED program designed to provide more rapid defibrillation to patients suffering life threatening dysrhythmias. Additional paramedics were hired and educated for each ambulance and each engine. A 12-lead ECG program was developed and implemented to reduce emergency department "door-to-drug" time in AMI. Quality improvement feedback mechanisms will assess effectiveness of the program and monitor scene times.

In the future, prevention will be an important focus (in addition to measuring effectiveness of the chain of survival and prehospital care for chest pain patients). Strategies that emphasize prevention include targeting at-risk patients, educating their families, making follow-up calls on chest pain patients who were transported via ambulance to emphasize risk factor education, and offering DFRS programs such as CPR. Educational efforts in the schools should be designed to teach students how to use the 911 system properly, and to educate them about diet, healthy lifestyle, and risk factors. This should be an area of focus for cardiac prevention programs. The goal of all these changes is to further reduce mortality and morbidity from ischemic heart disease in Howard County, Maryland.

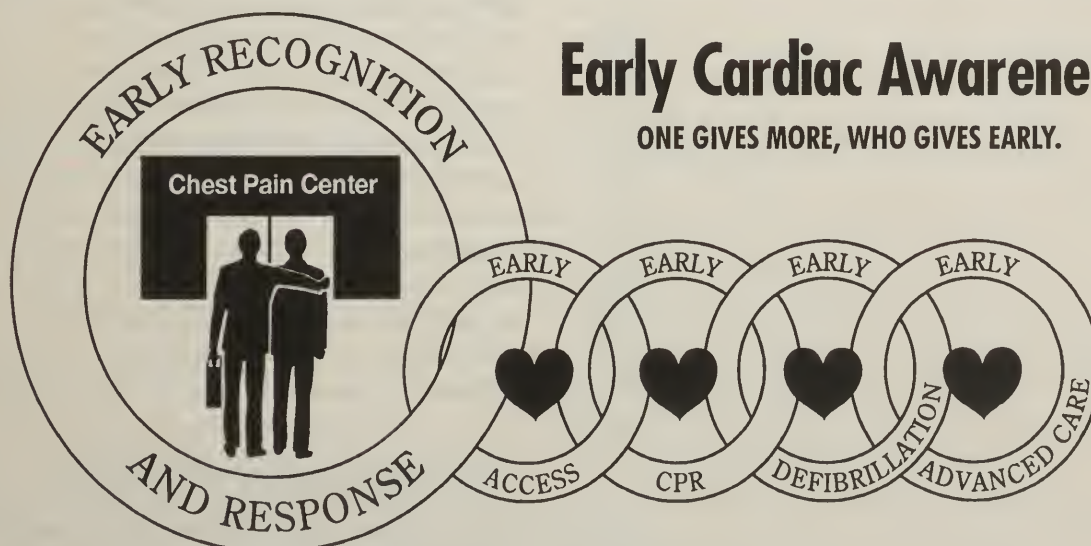
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Primary angioplasty in the treatment of acute myocardial infarction

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ABSTRACT: *There are more than 600,000 acute myocardial infarctions (AMIs) in the United States each year, with direct medical costs exceeding \$16 billion per year. Two treatment strategies are available for AMI today: medical therapy, including thrombolytic therapy, and primary angioplasty. Despite provocative preliminary data suggesting primary angioplasty results in lower mortality, morbidity and cost compared with thrombolytic therapy, most observers caution that more information is required before primary angioplasty replaces thrombolytic therapy for the treatment of AMI.*

Although significant strides in the diagnosis and management of coronary artery disease have been made in the last half of the 20th century, acute myocardial infarction (AMI) remains the single leading cause of death and disability in the United States.¹ While often considered a disease of the elderly, 45% of AMIs occur in persons less than 65 years of age.¹ The toll in human suffering due to death and disability and the economic cost to the individual and society are great because so many are affected.

The electrocardiogram (ECG) and clinical history at presentation can be used to categorize patients with AMI into three recognized groups.² Based on ECG at presentation, patients may be categorized as those with ST-segment elevation and those without ST-segment elevation. Based on clinical history, AMI patients with ST-segment elevation may be further divided into those eligible to receive thrombolytic therapy and those ineligible for thrombolytic therapy because of a contraindication to its application.

Therapy for acute myocardial infarction

Medical therapy. AMI patients presenting with ST-segment elevation typically have thrombotic occlusion of an atherosclerotic coronary artery. In these patients, thrombolytic therapy reduces mortality when administered within 12 hours of the onset of symptoms.³ The benefit of throm-

bolytic therapy is related to the time between symptom onset and application of therapy, with an estimated 39, 30, 26, and 20 lives saved per 1,000 treated patients when therapy is applied an average of 1, 3, 5, and 9 hours after symptom onset, respectively.³ Whether thrombolytic therapy is beneficial when administered more than 12 hours after symptom onset is less clear.³⁻⁵ In addition to thrombolytic therapy, medical therapy in these patients includes aspirin, analgesia, nitrates, beta-blockers, ACE inhibitors, oxygen, and heparin.²

Currently, most patients presenting within 12 hours of onset of AMI associated with ST-segment elevation receive thrombolytic therapy.⁶ Those who do not are considered thrombolytic ineligible for one or more reasons including uncontrolled severe hypertension, stroke, recent surgery, or a risk of bleeding.⁷ Thirty-day mortality in those considered ineligible for thrombolysis is significantly higher than in thrombolytic-eligible patients, with in-hospital mortality reported to be between 14%³ and as high as 33% in some patients (eg., those with a stroke or other bleeding risk) who often have multiple contraindications to thrombolytic administration.⁷ For this group, early medical therapy may include aspirin, analgesia, nitrates, beta-blockers, ACE inhibitors, oxygen, and, in certain cases, heparin (eg., large anterior infarction or demonstrated left ventricular thrombus on echocardiography).²

In AMI patients presenting without ST-segment elevation, significant coronary atherosclerosis is usually present, but complete thrombotic occlusion of the infarct-related artery is less common than in patients with ST-segment elevation.⁸ As a group, these patients do not benefit from thrombolytic therapy.^{3,9} In the emergency room, identifying individuals who actually have coronary artery disease and are at high risk for death or myocardial infarction among those presenting with chest pain but without ST-segment elevation is often difficult. In the Thrombolysis in Myocardial Ischemia (TIMI) IIIB Trial, a combination of ECG at presentation and clinical history (60 minutes of chest pain, ST-depression on ECG, no history of angioplasty, new angina) could be used to identify individuals who went on to have a myocardial infarction.¹⁰ While very specific, the sensitivity of this algorithm was quite poor.¹⁰ Recent evidence suggests that a combination of ECG at presentation and troponin level distinguishes low-risk from high-risk patients. Thus, patients with T-wave inversion only on presenting ECG represent a low-risk group,¹¹ even if troponin level is elevated;¹² mortality in this group is reported to be between 2% and 5%. However, patients who present with chest pain, ST-segment depression on ECG and an elevated troponin level have a mortality close to 12%.¹² Thirty-day mortality in AMI patients without ST-segment elevation on presenting ECG has been reported to be 11.6%,¹² 13.8%,³ and as high as 18%.¹³ Medical therapy includes aspirin, nitrates, beta-blockers, oxygen, ACE inhibitors, and heparin.²

Primary angioplasty. Recently, primary angioplasty has emerged as an alternative treatment for patients with AMI. Primary angioplasty is the mechanical reopening of an occluded vessel using a balloon-tipped catheter in patients with AMI who have *not* received antecedent thrombolytic therapy.

Patients with ST-segment elevation infarction who are thrombolytic eligible: Primary angioplasty has been compared to streptokinase and tissue plasminogen activator (t-PA) for treatment of AMI in five prospective, randomized trials involving thrombolytic-eligible patients presenting with ST-segment elevation.¹⁴⁻¹⁸

Two of these trials,^{14,16} with a combined sample size of approximately 800 patients, show, in meta-analysis¹⁹ a $\geq 50\%$ reduction of in-hospital mortality and reinfarction with primary angioplasty compared with thrombolytic therapy. The mortality benefit alone is 30 *additional* lives saved per 1,000 treated patients. The recent GUSTO IIb trial compared primary angioplasty with t-PA in 1,200 patients but found no difference in mortality or reinfarction.¹⁸ While there was a 30% reduction (13.7% to 9.6%, $p=0.033$) in the *composite* endpoint of death, recurrent infarction, and disabling stroke at 30 days in patients treated with primary angioplasty (initially reported as a 23% reduction, $p=0.06$, in abstract form),¹⁸ even this benefit was lost at six months. The two other randomized trials^{15,17} were quite small (100 patients in each) and were neither sized for nor detected any difference in mortality, reinfarction or stroke for patients treated with thrombolytic therapy or primary angioplasty. A meta-analysis that includes all randomized trials has not been published at this writing.

The Myocardial Infarction Triage and Intervention (MITI) Project Registry, while not a randomized trial, is the largest study published to date comparing primary angioplasty with thrombolytic therapy in AMI patients: 1,050 underwent primary angioplasty and 2,095 were treated with thrombolytic therapy.²⁰ Both high volume tertiary hospitals and lower volume community hospitals were included among the 19 participating centers. There was no difference in in-hospital (approximately 5.5%) or three-year (approximately 15%) mortality between patients treated with primary angioplasty and those treated with thrombolytic therapy, even after controlling for differences in baseline characteristics known to affect mortality.²⁰ The disparity between this study and the randomized trials could be due to the nonrandomized design; a lower incidence of stroke and faster application of thrombolytic therapy have also been suggested as potential explanations.²⁰ It is also possible that primary angioplasty is not as effective when applied in the community as when applied by highly motivated, experienced operators working in the high-volume centers that typically participate in randomized clinical trials.

Other patient groups. There is one preliminary report of a 197-patient randomized trial comparing a strategy of immedi-

ate angiography followed by angioplasty, if appropriate, to medical therapy in patients considered ineligible for thrombolytic administration.²¹ Patients were considered ineligible primarily because of a nondiagnostic ECG, although some presented late (≥ 6 hours) in the course of AMI. There was no difference in mortality or infarction rate in patients treated with the invasive strategy or with medicine alone, although the incidence of recurrent ischemia was reduced in the invasive group.²¹

Except for this one preliminary report, there are no randomized, prospective studies comparing primary angioplasty with medical therapy for treatment of patients who are ineligible for thrombolytic therapy, whether because of a contraindication to its administration or because ST-segment elevation is not present on the initial ECG. As noted above, these are high-risk AMI patients. In-hospital mortality of patients with AMI considered thrombolytic ineligible and treated with primary angioplasty is also high and is reported between 9% and 14%.^{21,22}

Time to treatment. Like thrombolytic therapy, the benefits of primary angioplasty depend upon its prompt application. In the randomized trials reported above, the time between arrival in the emergency room to balloon inflation (door-to-balloon time) averaged 90 minutes and was almost always less than 120 minutes. Several studies suggest a clear relation between poor outcomes and prolonged door-to-balloon time;²³⁻²⁶ mortality may rise as much as 50% when door-to-balloon time exceeds 120 minutes.²⁵

Primary angioplasty and on-site cardiac surgery. In accordance with American College of Cardiology/American Heart Association (ACC/AHA) Task Force guidelines,²⁷ most state health plans prohibit or strongly discourage performance of angioplasty at hospitals without on-site cardiac surgery. Since the majority of patients with AMI present to hospitals without an on-site cardiac surgical program and, therefore, without an angioplasty program, access to intervention-based reperfusion therapy is denied.

But is on-site cardiac surgery necessary for *primary* angioplasty? The weight of evidence suggests the answer is no. No patient was taken for emergency bypass surgery *as a result of an angioplasty complication* in any published, prospective randomized trial comparing primary angioplasty with thrombolytic therapy in the treatment of AMI¹⁴⁻¹⁷ or in the largest, published consecutive series.²² This is not surprising since the event that necessitates emergent coronary artery surgery is actual or threatened vessel occlusion. In AMI coronary occlusion has already occurred. And while a non-infarct-related artery can be injured in the course of angioplasty, this is extremely uncommon. Still, approximately 5% of AMI patients for whom primary angioplasty is the intended treatment require coronary artery bypass surgery because of anatomy unsuitable for angioplasty¹⁶ or, less frequently because

angioplasty fails to recanalize the vessel. If coronary artery surgery is required, patients can be safely transported to a cardiac surgical center.²⁸

Despite these considerations, data concerning primary angioplasty at hospitals *without* cardiac surgical programs demonstrate safety and efficacy in some series,²⁹ but not all.^{23,24} Less than optimal outcomes in such centers can be related to prolonged door-to-balloon times, but other potential reasons include low-volume operators and laboratories, or preprocedure and postprocedure caregivers inexperienced with triage and care of patients undergoing primary angioplasty.

The conclusion from these considerations is that primary angioplasty at hospitals without on-site cardiac surgical programs can be safe and effective if the primary angioplasty program is properly developed and assures the prompt application of therapy by knowledgeable caregivers. How programs are best developed and implemented has been neither formalized nor studied in any rigorous way, and yet is crucial to the safe and effective application of this therapeutic alternative.

Economic aspects of acute myocardial infarction

The high cost of coronary artery disease is a reflection of its prevalence, and its killing and disabling of persons, often in their most productive years. In 1989, coronary artery disease cost an estimated \$22 billion in medical care costs and \$32 billion in indirect costs.³⁰ Acute myocardial infarction represents a large part of this cost since its incidence is high and its treatment, expensive. Applying the recent Mayo Clinic data³¹ to the U.S. AMI population suggests medical costs for AMI exceed \$16 billion per year.

Data concerning comparative costs of primary angioplasty and medical therapy are limited, conflicting, and involve only thrombolytic-eligible patients; often charges, not costs, are reported. In the 100-patient Mayo Clinic study, while no statistically significant differences were observed, primary angioplasty tended to be less expensive than thrombolytic therapy. Hospital charges for AMI were \$26,700 for t-PA and \$21,000 for primary angioplasty ($p=0.09$); 12-month charges were an additional \$3,600 and \$1,200 for t-PA and primary angioplasty, respectively (P value not significant).³¹ Hospital stay was shorter by two days and return to work sooner by 18 days in the primary angioplasty group.³¹ In-hospital charges for patients enrolled in the Primary Angioplasty in Myocardial Infarction (PAMI) study were approximately 12% less in patients undergoing primary angioplasty compared with those treated with t-PA.³² In contrast, for patients enrolled in the GUSTO II trial, six-month direct medical costs of primary angioplasty and of t-PA were similar (\$18,643 and \$19,395, respectively).³³ And finally, the largest but nonrandomized comparison from the MITI Project Regis-

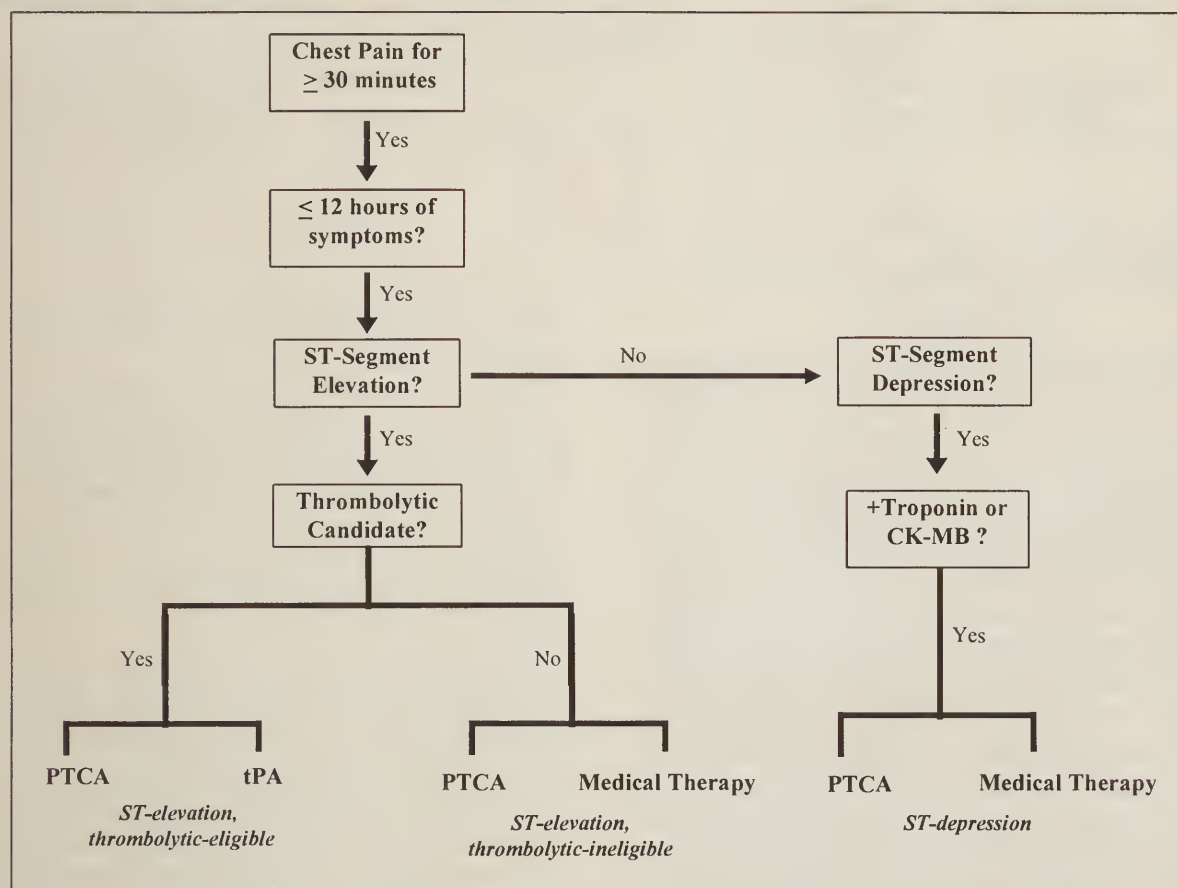


Figure 1. Schematic outline of the C-PORT trial.

try showed that hospital cost was lower ($p < 0.001$) with thrombolytic therapy (\$16,838) than with primary angioplasty (\$19,702).²⁰

Summary and the need for further study

These considerations suggest that the place of primary angioplasty in the treatment of AMI remains unclear and controversial, even in thrombolytic-eligible patients with ST-segment elevation infarction. In this best-studied group, whether the relative advantage of primary angioplasty is very large,^{14,16,19} more modest,¹⁸ or even exists²⁰ is not clear. Importantly, whether differences in outcome among reported trials are related to where or by whom primary angioplasty is performed, is also unknown. In the higher risk patient groups — those with ST-segment elevation who are ineligible for thrombolysis and those with ST-segment depression at presentation — there are few or no comparative medical outcomes data.

Economic data comparing primary angioplasty and thrombolytic therapy are limited and are primarily direct medical cost data in thrombolytic-eligible patients. In order to resolve the important economic issues a study must have an adequate sample size and a comprehensive summary of all direct and indirect costs. Finally, the effect of

these alternative therapies on quality of life remains unstudied in any patient group.

Important questions of science and of policy remain unanswered. Continued uncertainty about which therapy is better from a medical, economic, or quality-of-life point of view mandates further study, particularly since there is suggestive, but not unequivocal, evidence that one therapy may be significantly better than the other. At the same time, the issue of whether primary angioplasty should be developed at community hospitals without on-site cardiac surgical programs is a matter of intense interest and controversy. These two issues, the relative benefit of primary angioplasty in the treatment of AMI and access to primary angioplasty at hospitals without on-site cardiac surgery, are inextricably bound. To define the relative benefit of primary angioplasty in the "real-world" practice of cardiology, a clinical trial must be performed in that real-world setting. And whether primary angioplasty programs should be developed at community hospitals around the nation importantly depends upon the relative benefit of primary angioplasty in the treatment of AMI. Because the issue of relative benefit remains unsettled, and while the number of primary angioplasty programs is relatively small, there is now a window of opportunity to implement a clinical trial designed to address both issues.

TABLE 1. C-PORT clinical trial outcomes in first 70 patients.

	Chest Pain to ED (min)	ED to Cath I/Lab (min)	ED to Inflation (min)	Pre-PTCA % Stenosis	Post-PTCA %Stenosis	Pre-PTCA TIMI Flow	Post-PTCA TIMI FLOW
Mean	142	67	103	98	12	0.97	2.4
Median	86	67	98	100	10	0	3
Range	31-690	15-150	60-192	90-100	0-40	0-3	1-3

ED, emergency department; PTCA, percutaneous transluminal coronary angioplasty.

The C-PORT trial

The Atlantic Cardiovascular Patient Outcomes Research Team (C-PORT) is a group of cardiovascular specialists (including physicians, nurses, and technicians), health care economists, quality-of-life researchers, clinical trials specialists, and biostatisticians who have come together for the purpose of designing, developing, and conducting community-based, clinical cardiovascular trials. This group has developed a trial comparing primary angioplasty with medical therapy in patients presenting with acute myocardial infarction. While previous randomized trials conducted primarily in academic or tertiary centers involved only patients presenting with ST-segment elevation infarction who were thrombolytic candidates, the proposed trial extends the comparison to AMI patients with ST-segment elevation considered thrombolytic-ineligible and to those presenting with ST-segment depression. A schematic outline of the C-PORT trial is shown in **Figure 1**.

The C-PORT trial includes a wide range of treatment facilities and practitioner practice styles and uses widely accepted guidelines to determine practitioner and facility participation, reducing selection biases which would otherwise limit the applicability of trial results. For example, those performing angioplasty in the clinical trial must meet the ACC/AHA criteria for maintenance of competency (75 cases per year). The C-PORT trial provides for a comprehensive assessment of outcomes including morbidity and mortality, comparative costs, and quality of life. The trial has begun in Maryland and currently involves five community hospitals in the Baltimore-Washington area. It will begin shortly in Massachusetts, Pennsylvania, New Jersey, and Rhode Island, ultimately involving 50 sites.

Many hospitals involved in this trial have on-site cardiac surgery and angioplasty programs, but many do not. To implement the proposed clinical trial, primary angioplasty programs must be established at hospitals which currently do not perform this procedure. To do this, a formal primary angioplasty development program has been established which addresses the many training, logistical, and quality management issues involved in performing primary angioplasty at a community hospital without on-site cardiac surgery. This C-PORT development program may serve as a model for establishing primary angioplasty in other communities, should trial

results demonstrate that greater access to this procedure is desirable.

The C-PORT trial will not report primary outcomes (death, recurrent infarction, stroke) until trial completion. However, there are interesting early results related to the ability of community hospitals to apply primary angioplasty in a prompt and successful fashion. Outcomes in the first 70 patients entered into the C-PORT clinical trial at five community hospitals in the Baltimore-Washington area are reported in **Table 1**.

These data show that primary angioplasty can be applied quickly and effectively in community hospitals. It is important to note that a formal angioplasty development program was applied in each of these institutions prior to study initiation. This program included training of catheterization laboratory nursing and technical staff, and critical care unit and emergency room staff (ie., didactic sessions, observational training at tertiary facilities, supervised "dry runs") and local logistics development.

The implications of this trial for future health care policy and planning are great. As with trauma in the 1960s, there is growing awareness that an emergency medical system (EMS) must be in place for the proper triage of the more than five million people presenting to U.S. emergency rooms each year with chest pain of possible cardiac origin. The C-PORT trial will determine if primary angioplasty is superior to medical therapy for treatment of AMI; and, if it is better, *in what hospitals, by whom, and how* it should be performed. Results from this trial will not define the detailed structure of an effective EMS for AMI, but it *will* define what it needs to do. Policymakers and planners can look to this trial for the standards, logistical goals and techniques, and quality management strategies required to develop primary angioplasty at community hospitals without on-site cardiac surgery, if trial results support the need for establishing such programs. On the other hand, if primary angioplasty is no better than medical therapy, then the need for their nationwide development would be greatly lessened.

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A path forward for chest pain care: what are the expectations of each team member?

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ABSTRACT: *It would be impossible to expect that an immediate solution to the heart attack problem in the United States can come out of this multidisciplinary conference. A more practical goal would be the conferees coming together, seeing better what needs to be done, and using interpretative abilities to put together a logical approach which would unite both the community hospital and the community by the first step on the learning curve. Future progress will come about from the multidisciplinary team with research studies and a commitment to solving this problem on a local level. Acting locally and thinking globally will enable us to move forth and find the expectations of each member of the team. The challenge will find us many times in the years ahead.*

The First Maryland Chest Pain Center Research Conference, for the first time ever, successfully challenged the notion that a cooperation among hospitals within a state could become part of a learning curve of progress to take heart attack deaths out of first place. Heart disease remains the number one killer of the adult population across the United States. This conference challenged the health care industry of Maryland to go beyond present-day knowledge and put together a strategy to achieve victory with this altruistic effort. It blended evidence-based medicine with interpretive medicine as defined by Richard Horton, M.D., editor of *Lancet*,¹ to accomplish this.

The learning curve has become a tool for progress, as seen with the development of widespread coronary care units throughout the United States. Unfortunately, the “zenith” of the coronary care curve seems to be coming to a point of diminishing return. Medical therapy has improved tremendously, but so far has not seemed adequate to reach the people who most need early therapy. The barrier to this appears to be self-imposed. To overcome this barrier, the paradigm shift to understanding and utilizing heart attack beginnings stands out. Evidence-based medicine tells us that approximately 50% of patients with heart attacks have prodromal symptoms. In the GUSTO II (Global Use of Strategies to Open Occluded

Immediate Goals:

1. To reduce time to treatment after hospital arrival for myocardial infarction patients.
2. To reduce the prehospital delay of myocardial infarction patients by educating them to seek help early (change behavior).
3. To achieve prevention of heart attack by early intervention (early heart attack care/EHAC), i.e., to convert late presentation of myocardial infarction to earlier (prodromal stage) presentation, by changing patient behavior.

Figure 1. Chest Pain Centers in EDs: Damage Control Centers.

Long-Range Goal:

To reduce the time it takes to eliminate heart attack deaths (or reduce it from its number one position) by the year 2000. The plan to accomplish this is widespread community education focused on EHAC coupled with a widespread hospital chest pain emergency department programs.

Figure 2. Chest Pain Centers in EDs: Damage Control Centers.

Arteries) study, the median for these prodromal symptoms was two days.² Symptoms at this prodromal stage are perceived differently in that they are not always painful and prolonged in presentation. The mildness and intermittency, as well as lack of awareness of their importance, contributes to the problem. Spending time, effort, and energy in this direction may prove more beneficial in the long run than additional information on thrombolytic agents and other therapies.

In association with the awareness of heart attack beginnings, the learning curve for chest pain center development has taught us that the low probability ischemic disease pathway is an excellent way to sort out patients with mild intermittent chest symptoms. Eighty percent of such patients can be safely discharged home from the emergency department, thus opening the door for patients presenting with early prodromal symptoms (Figures 1, 2). What is now needed is an understanding and an effort to move in the direction of early action and this is what the early heart attack care (EHAC)

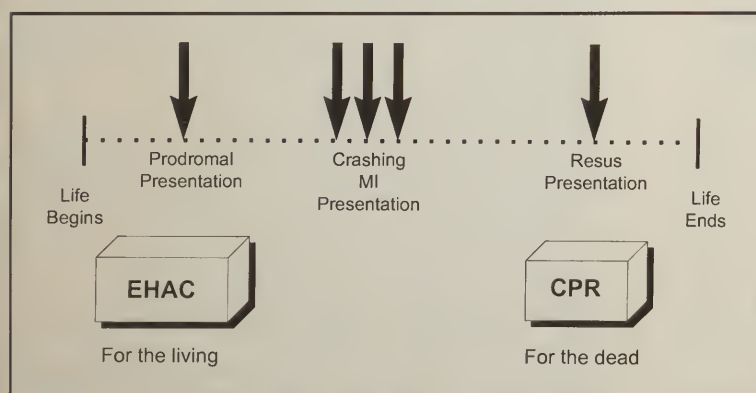


Figure 3. Spectrum of Ischemic Heart Disease.

$$\text{CRASHING INDEX} = \frac{A + B}{A + B + C}$$

Myocardial Infarction Index

- ✓ Failure to Detect
- ✓ Missed Signs
- ✓ Lost Opportunities

A = MI + Thrombolysis

B = MI + No Thrombolysis

C = Unstable Angina

Figure 4. Myocardial Infarction Index.

movement is all about — shifting the paradigm of care to acute prevention of a heart attack (Figure 3).

What is this higher level of performance for a community hospital? Guidelines for the management of acute myocardial infarction and unstable angina are available from the American College of Cardiology and the American Heart Association. However, implementation has been a problem. The National Heart Attack Alert Program (National Institutes of Health) attempts to implement the guidelines on a national level by focusing on reduction in treatment time for patients with crashing acute myocardial infarction once they hit the emergency department.³ These patients are now expected to be treated within 30 minutes. It is important to remember that reduction of time within the hospital is only important if the time outside the hospital is also reduced significantly. Gaining ten minutes in the emergency department when the patient has waited over three hours in the community becomes less important. The focus needs to broaden and embrace time reduction in the community — the overall time from onset to treatment approaches the golden first hour for maximum benefits to take place. Not only is there a reduction here in mortality to 1% in patients treated in under one hour, but acute prevention takes place in 40% of the patients who have their heart damage aborted. Thus performance measurements in hospitals need to embrace not only “door-to-needle time” but also the total time from onset of symptoms to therapy, which becomes the real determinant of morbidity and mortality in heart attack patients.⁴

However, in the future, hospital performance needs to be measured for total acute ischemic experience. This will involve patients not only with ST elevated acute myocardial infarction (Group “A”), and non Q-wave infarction (Group “B”), but also patients with various forms of unstable angina (Group “C”). It is the latter category that we should be encouraging and promoting to enter the emergency department because it is here where acute prevention can have its highest benefits (Figure 4). We can postulate an index entitled “The Early Index” or the “Acute Pre-

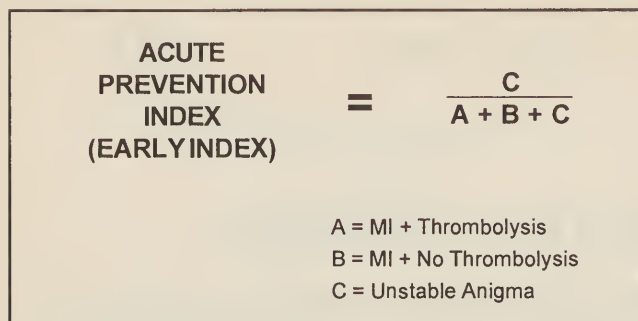


Figure 5. Acute Prevention Index.

vention Index” which is $C/A+B+C\%$ (Figure 5). The reverse of this would be the “Myocardial Infarction Index” or the “The Failure to Detect Index,” which equates to missed signs and lost opportunities and equals $A+B/A+B+C\%$ (Figure 4). Patients in group “C” represent a group presenting as unstable angina who can be prevented from having acute myocardial infarction. To this group we could also add “A,” which measures ST-elevated acute myocardial infarction patients being treated within the first hour where acute prevention is also seen. Thus acute prevention or early index could be better defined as $C+A^1/A+B+C\%$ (Figure 6). When this is carried out then all patients presenting with acute myocardial ischemia can be entered into the acute ischemic equation and a hospital’s performance measured by the percentage of patients in each category.⁵ These results could be compared to other hospitals in such a way as to have hospitals compete for optimal heart attack performance. Along these lines it is wise to remember the words of William Kannel, M.D., professor of medicine public health at Boston University, who stated, “the day has to come when we consider heart attack in our patients as not the first indication of treatment, but as a medical failure.” He may have been speaking of prevention of heart attack in regards to primary risk factor reduction, but acute heart attack prevention best demonstrated at the prodromal unstable angina stage, rather than the thrombolytic stage, can now be added to this prophetic statement.

Every physician should evaluate his or her institution and determine what the plan is for dealing with the heart attack problem within the community. The plan should include the use of a team effort and a continuous quality improvement process. Championing the cause to address the problem does

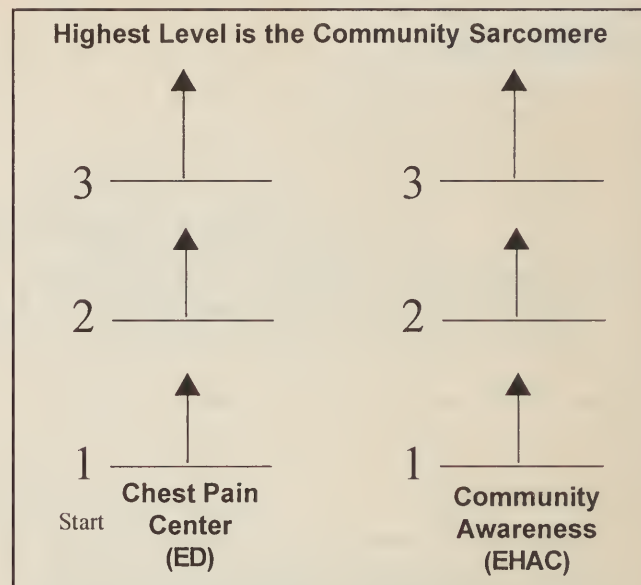


Figure 7. Levels of Performance.

not depend on physician specialty; all that is required is a commitment to understand what is needed to move in the proper direction and to act accordingly. If a physician pictures himself or herself on that learning curve at Point 1 and wants to go to Point 2 over the next several years (Figure 7), he or she must discover how to bring members of the team together. That is the real challenge. Once this challenge is accepted, physicians become believers in themselves and in their hospital’s attempt to do so and in the overall strategy to reduce significantly heart attack deaths within the country. The expectation is to move ahead and understand that no difficult task is easy and that each physician is among many others who embrace the same mission. The learning curve enables you to focus clearly on the problem and a togetherness that allows you to overcome seemingly resistant points along the way, helping you to expand the frontiers of your minds. Creativity thus becomes just as important as facts. What can be imagined can be created. Heart disease is vulnerable and can be removed from first place as our nation’s number one heart problem.⁶

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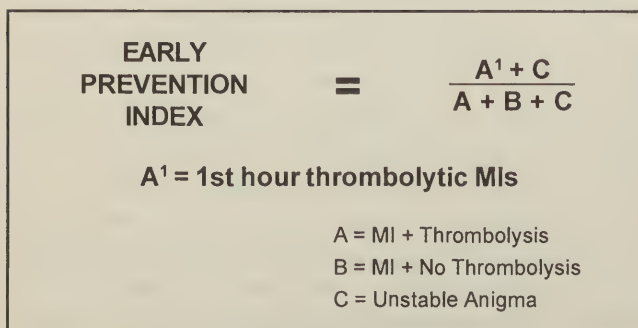
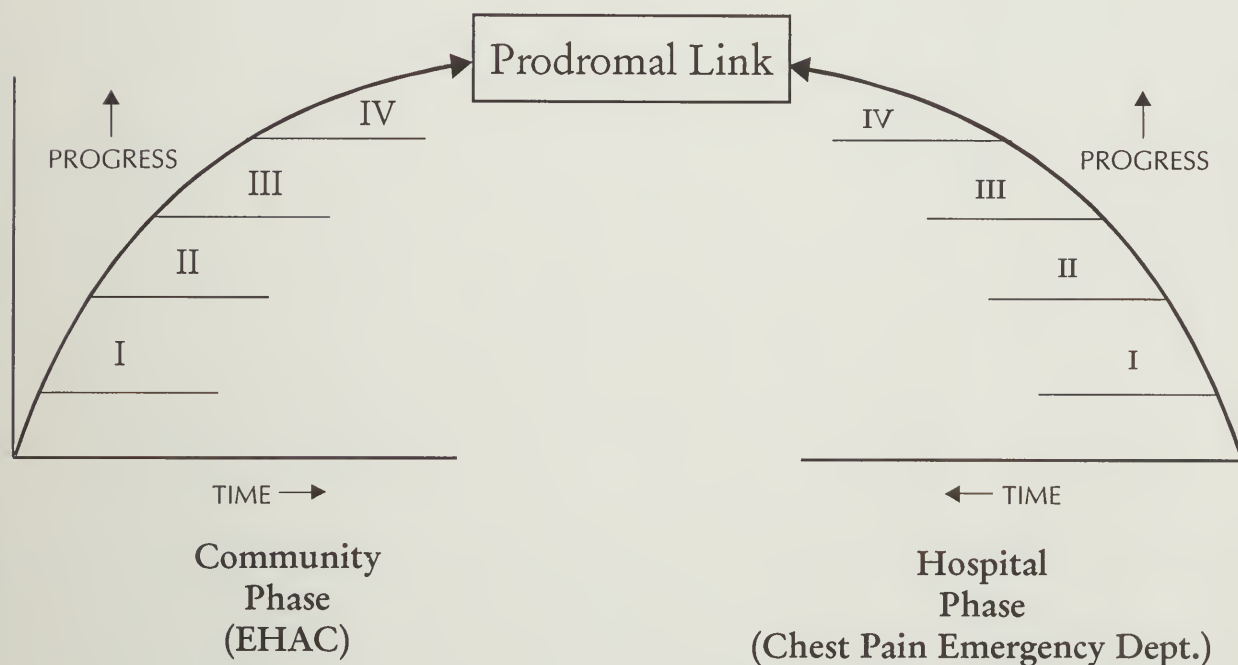


Figure 6. Early Prevention Index.

THE COMMUNITY SARCOMERE

Strategy for Effective Community Penetration



COMMUNITY PHASE

- IV ---EHAC specific groups bringing about widespread community penetration
- III ---The EHAC Message at the other end of the spectrum from CPR
- II-----Cardiac Outreach Program
- I -----Bedside message learned in CCU rounds

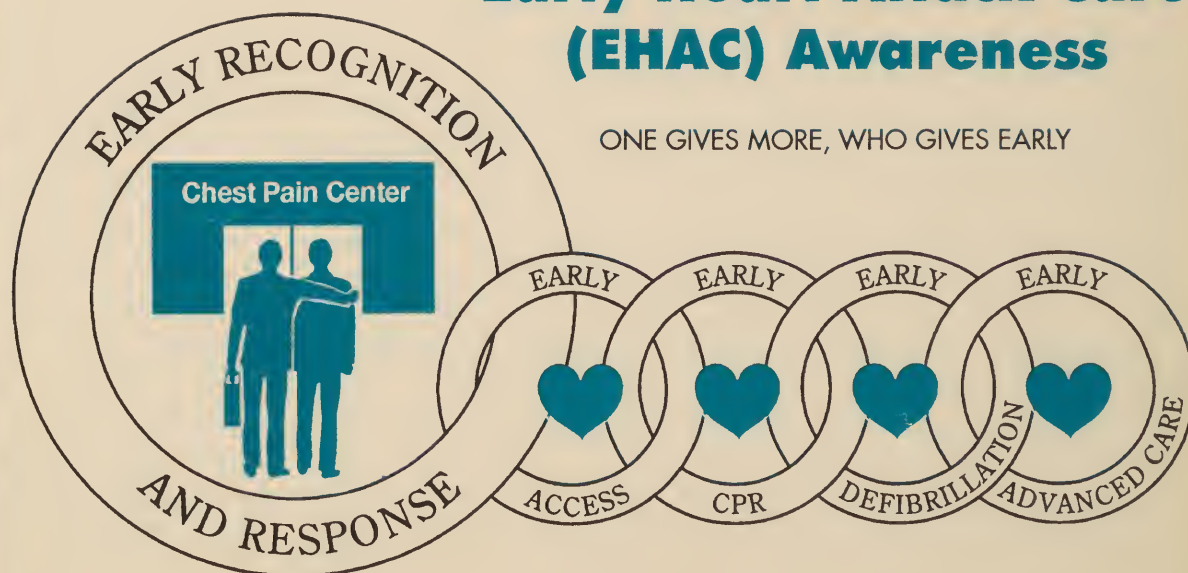
HOSPITAL PHASE

- IV ---Perfection of the low probability ischemic pathway. The use of platelet IIb/IIIa receptor antagonists.
- III ---The Development of critical pathways (Tracks, levels, etc.)
- II-----Comprehensive, systematic management of chest pain patients
- I -----Emphasis on chest pain patients as a way to get the heart attack problem



Early Heart Attack Care (EHAC) Awareness

ONE GIVES MORE, WHO GIVES EARLY



Chest Pain Centers: Management of Central Chest Pain

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